VOLUME 7 *Chicago Number* NUMBER 2

THE MEDICAL CLINICS OF NORTH AMERICA

SEPTEMBER, 1923

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

COPYRIGHT, 1923, W. B. SAUNDERS COMPANY. ALL RIGHTS RESERVED

PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA

MADE IN U. S. A.

MEDICAL LIBRARY
JUN 5 19
UNIV. OF N. DAKOTA

CONTRIBUTORS TO THIS NUMBER

ISAAC A. ABT, M. D., Professor of Diseases of Children, Northwestern University Medical School; Attending Physician, Diseases of Children, Sarah Morris Children's Hospital and Michael Reese Hospital.

PETER BASSOE, M. D., Professor of Medicine (Nervous and Mental Diseases), Rush Medical College; Attending Neurologist, Presbyterian Hospital, Grant Hospital, Evanston Hospital, Home for Destitute Crippled Children.

ARTHUR F. BYFIELD, M. D., Assistant Professor of Medicine, Rush Medical College; Attending Staff (Medicine), Cook County Hospital.

JAMES GRAY CARR, M. D., Associate Professor of Medicine, Northwestern University Medical School; Attending Physician, Cook County and Wesley Memorial Hospitals.

PHIL A. DALY, M. D., Associate Attending Physician, Chicago Memorial Hospital; Chicago Lying-in Hospital (Internal Medicine); Clinical Instructor in Medicine, Northwestern University Medical School.

NATHAN S. DAVIS, III, M. D., Associate in Medicine, Northwestern University Medical School.

CHARLES A. ELLIOTT, M. D., Professor of Medicine, Northwestern University Medical School; Attending Physician, Wesley Memorial Hospital.

J. C. FRIEDMAN, M. D., Attending Physician, Michael Reese Hospital.

JESSE R. GERSTLEY, M. D., Instructor in Pediatrics, Northwestern University Medical School; Associate Attending Pediatrician, Michael Reese Hospital.

J. P. GREENHILL, M. D., Demonstrator in Operative Obstetrics, Northwestern University Medical School.

CLIFFORD G. GRULEE, M. D., Associate Professor and Acting Head of the Department of Pediatrics at Rush Medical College (in affiliation with the Univeristy of Chicago); Chief of Pediatric Staff, Presbyterian Hospital.

WALTER W. HAMBURGER, M. D., Assistant Professor of Medicine, Rush Medical College; Associate Attending Physician, Michael Reese Hospital.

RALPH C. HAMILL, M. D., Assistant Professor of Neurology, Northwestern University Medical School; Attending Neurologist, Wesley Memorial Hospital; Consulting Neurologist, United States Public Health Service, Chicago.

JULIUS H. HESS, M. D., Professor and Head of the Division of Pediatrics, University of Illinois College of Medicine; Chief of Pediatric Staff, Cook County Hospital; Attending Pediatrician to Cook County, Michael Reese, and Englewood Hospitals; Consulting Pediatrician, Municipal Contagious Hospital, Chicago; Member of the Advisory Committee to the Children's Bureau, Department of Labor, Washington, D. C.

CHARLES LOUIS MIX, M. D., Professor of Medicine and Head of the Department, Loyola University Medical School.

WALTER H. NADLER, M. D., Associate in Medicine, Northwestern University Medical School.

VINCENT J. O'CONOR, M. D., Urological Surgeon to the Washington Boulevard Hospital; Instructor in Genito-urinary Surgery, University of Illinois College of Medicine.

MILTON M. PORTIS, M. D., Chicago.

ROBERT SONNENSCHEIN, M. D., Professor of Otolaryngology, Post-Graduate Medical School; Assistant Professor of Otolaryngology, Rush Medical College; Consulting Otolaryngologist, Durand Hospital of the McCormick Institute for Infectious Diseases; Associate Attending Otolaryngologist, Michael Reese Hospital.

SOLOMON STROUSE, M. D., Associate Attending Physician, Michael Reese Hospital; Attending Physician, Chicago Lying-in (Internal Medicine), Chicago Memorial Hospital; Associate Professor of Medicine, Northwestern University Medical School.

DON C. SUTTON, M. D., Assistant Professor of Medicine, Northwestern University Medical School; Attending Physician, Cook County Hospital.

FREDERICK TICE, M. D., Professor of Medicine, College of Medicine, University of Illinois; Attending Physician, Cook County Hospital.

CHARLES SPENCER WILLIAMSON, M. D., Professor and Head of the Department of Medicine, University of Illinois; Attending Physician, Cook County Hospital.

3163

CONTENTS

PAGE

Clinic of Dr. Isaac A. Abt, *Michael Reese Hospital*
THE NATURE AND TREATMENT OF COLLAPSE IN INFANCY AND CHILDHOOD 317

Clinic of Dr. Charles Louis Mix, *Mercy Hospital*
DUODENAL ULCER COMBINED WITH CHOLECYSTITIS 337

Clinic of Drs. Charles A. Elliott and Walter H. Nadler, *Wesley Memorial Hospital, Northwestern University Medical School*
CHRONIC GALL-BLADDER DISEASE 349

Clinic of Dr. Charles Spencer Williamson, *Cook County Hospital*
ACUTE LYMPHATIC LEUKEMIA (SMALL-CELLED TYPE) 369
PURPURA HÆMORRHAGICA 379
HODGKIN'S DISEASE (ABDOMINAL TYPE) 387

Clinic of Dr. J. C. Friedman, *Michael Reese Hospital*
LATENT JAUNDICE 397

Clinic of Dr. Ralph C. Hamill, *Children's Memorial Hospital*
INFANTILE CEREBRAL PALSIES 407

Clinic of Drs. Frederick Tice and Vincent J. O'Conor, *Washington Boulevard Hospital*
DISEASE OF THE UPPER URINARY TRACT WITHOUT REFERABLE SYMPTOMS 429

Clinic of Dr. Vincent J. O'Conor, *Washington Boulevard Hospital*
POSSIBILITIES OF ERROR IN THE DIAGNOSIS OF RENAL CALCULI 437

Clinic of Dr. Milton M. Portis, *St. Luke's Hospital*
PEPTIC ULCER 449
GANGRENE OF THE TOES THE RESULT OF ENDARTERITIS OBLITERANS 455
THROMBOSIS OF THE VESSELS OF THE LEG AND THE MESENTERIC VESSELS 459

Clinic of Dr. Walter W. Hamburger, *Michael Reese Hospital*
ACUTE CARDIAC PSYCHOSES: ANALYSIS OF THE TOXIC AND CIRCULATORY FACTORS IN 5 CASES OF ACUTE CONFUSION 465

Clinic of Dr. Clifford G. Grulee, *Presbyterian Hospital*
THE CARE AND FEEDING OF PREMATURE INFANTS 477

Clinic of Drs. Solomon Strouse and Phil A. Daly, *Michael Reese Hospital*
THE INTERNIST AND THE SURGICAL ABDOMEN 487

Clinic of Dr. Julius H. Hess, *Cook County Hospital*
SOME CHRONIC ABDOMINAL CONDITIONS IN INFANCY AND CHILDHOOD. PRESENTATION OF THREE GROUPS OF CHRONIC ABDOMINAL CASES 499

Clinic of Dr. Peter Bassoe, *Presbyterian Hospital*
EPILEPSY WITH REPORT OF 200 CASES SEEN IN PRIVATE PRACTICE 519

Clinic of Dr. Robert Sonnenschein, *Postgraduate Hospital*
A GROUP OF NOSE AND THROAT CASES PRESENTING GENERAL SYSTEMIC SYMPTOMS 533

Clinic of Dr. Nathan Smith Davis, III, *Medical Dispensary Clinic, Northwestern University Medical School*
ARTERIAL HYPERTENSION: ITS RELATIONSHIP TO CHRONIC INTESTINAL DISORDERS 547

Clinic of Dr. Jesse R. Gerstley, *Northwestern University Medical School*
RESHAPING SOME IDEAS OF INFANTILE ECZEMA 555

Clinic of Dr. James G. Carr, *Cook County Hospital*
JAUNDICE 567
TUBERCULOSIS OF THE SEROUS MEMBRANES 583

Clinic of Dr. Arthur F. Byfield, *Cook County Hospital*
MEDICAL EMERGENCIES 597

Clinic of Dr. Don C. Sutton, *Cook County Hospital*
HAY-FEVER 605

Clinic of Dr. J. P. Greenhill, *Chicago Lying-in Hospital*
THE VALUE OF *x*-RAY IN OBSTETRICS 611

THE MEDICAL CLINICS
OF
NORTH AMERICA

Volume 7 Number 2

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL

THE NATURE AND TREATMENT OF COLLAPSE IN INFANCY AND CHILDHOOD

THE condition which we call collapse may be defined as a state of extreme prostration and depression with failure of the circulation. Consequently, we shall consider that collapse is due to circulatory failure.

In infancy and childhood the clinical picture of collapse varies greatly and appears in many conditions, and the manifestations of circulatory failure may resemble very closely those which occur in surgical shock. It is difficult to differentiate shock and collapse, but the term "shock" is usually reserved for the condition of depression and prostration which may follow surgical procedure, trauma, and accidental injury. Collapse, more often than shock, may be considered in relation to the infections and intoxications. Clinically and experimentally it is often impossible to distinguish shock from collapse.

Crile[1] attempts to distinguish between shock and collapse in stating that the onset of collapse is sudden and may have its origin in cardiac, respiratory, or vasomotor insufficiencies, while in shock the onset is gradual and is of vasomotor origin only. In both conditions there is a lowering of blood-pressure. In shock the vasomotor center is exhausted, while in collapse this center, when involved, is only functionally depressed. Collapse conditions tend to recover if stimulants are given, but in shock the response to stimulation is very slight.

W. H. Howell[2] in 1903 controverted Crile's theory by dis-

tinguishing two forms of shock—cardiac and vascular. He assumed that both varieties were due to an inhibition of the centers in the medulla, and that in cardiac shock there occurred an inhibition of the tonus of the vagus, with consequent acceleration of the heart. In vascular shock the vasomotor center is inhibited.

Howell's experiments were made on dogs anesthetized with morphin and ether and brought into a condition of shock by operations of various kinds. He reached the following conclusions:

1. The most important and dangerous feature of severe shock is a long-continued, practically permanent fall in blood-pressure to about 20 to 40 mm. of mercury. This condition is designated as vascular shock and is due to a sustained loss of activity of the vasoconstrictor center.

2. A second important result of shock is a very rapid and feeble heart-beat. This condition is designated as cardiac shock. Although it may result secondarily from the permanent fall in blood-pressure, it may also occur quite independently of the vascular shock as a primary result of the operations. Cardiac shock is due to a more or less permanent loss of activity of the cardio-inhibitory center.

3. Intravenous infusion with alkaline salt solutions (NaCl 0.6 per cent.—Na_2CO_3 0.5 per cent.) causes a rise in pressure by increasing the force of the heart-beat. The effect, Howell thought, was more durable than with salt solution alone and may be renewed by repeating the injection.

According to Howell, the fundamental cause of vascular and cardiac shock is not exhaustion of the vasomotor and cardio-inhibitory centers from overactivity, but a more or less permanent inhibition of these centers from excessive stimulation of the inhibitory paths.

Howell agrees with Crile that circulatory disturbance is the main factor in the production of shock, but he differs in finding that the cardio-inhibitory center participates in the causation of shock and does not concede that the vasomotor center has been exhausted.

It is evident as we proceed with our inquiry that, notwithstanding the attempts that have been made to differentiate shock from collapse, the two conditions are so closely related that in this discussion no further attempt will be made to separate them, and for practical purposes, at least, we shall consider the two processes as identical.

The experimental work and the clinical observations which have been reported deal almost exclusively with so-called shock and, as has already been said, we shall consider these two conditions as one process.

Collapse or shock may occur after severe hemorrhage or following surgical operations of any part of the body, particularly operations on the brain and the abdominal viscera. They may also occur where there is an obstruction to respiration or in acute cardiac infections. Collapse symptoms are frequently observed during the acute infectious diseases as well as in the disturbances of alimentation and nutrition of young infants.

Certain poisonous drugs, such as the corrosive agents, which produce eschars upon the mucous surfaces of the mouth, pharynx, esophagus, and remainder of the gastro-intestinal tract, may produce shock as a result of the irritation to the gastric branches of the vagus nerve which carry the stimuli to the ganglion cells of the medulla.

All drugs which depress the circulatory and nervous systems, such as cardiac drugs in large doses, coal-tar products, cyanids, hypnotics, narcotic drugs, nicotin, and most phenols, are capable of producing symptoms of collapse. The same is true of drugs which irritate the central nervous system. Among this group may be mentioned atropin, caffein, cocain, strychnin, and some of the volatile oils.

Young infants are extremely susceptible to extremes of heat or cold, and manifestations of severe collapse may occur if patients are unduly exposed to either extreme. This is particularly true of delicate or premature infants.

Children sometimes go into extreme collapse as a result of the so-called anaphylactic shock. This may occur when a

foreign serum, such as diphtheria or tetanus antitoxin or normal horse-serum, is administered hypodermically. It may also occur in those infants who show an idiosyncrasy to cow's milk and in whom the ingestion of a small quantity may produce alarming symptoms of prostration, with pallor and rapid, feeble pulse.

Anesthesia may cause sudden and alarming shock; indeed, a fatal termination may occur. Strong emotion, sudden fright or great fear, the infliction of so-called psychic trauma, may cause sudden, temporary, circulatory failure.

Nature of Shock or Collapse.—At the present day there are various hypotheses as to the nature and cause of shock or collapse conditions. The older clinicians thought that shock was due to some grave disturbance in the central nervous system, and considered it to be a "commotio cerebri" dependent upon molecular changes in the brain substance.

Recent physiologic investigations have shown that in shock or collapse there is a fall of blood-pressure and that the failure of the circulation in the infectious diseases may be ascribed to a damage of the vasomotor center and paralysis of the vasomotor nerves. It is maintained by some that the circulatory failure occurring in infectious diseases is the result of a collection of blood in the abdominal veins or capillaries and an insufficient supply of blood to the heart.

In substantiating this it may be stated that autopsies on children who have died from infectious disease show an accumulation of blood in the abdominal veins and capillaries. It is logical to assume that with such stagnation the blood-supply to the heart is diminished and insufficient. On the basis of the knowledge concerning surgical shock, and from additional observations, Hewlett suggests that the cause of circulatory collapse in severe infections is a stagnation of blood in the abdominal veins and capillaries.

There is still a great deal of confusion as to the exact mechanism of circulatory failure occurring in the infectious diseases. It does not seem definitely proved that either the heart or the vasomotor center is always the cause of death in those individuals who die with symptoms of circulatory failure. On the

other hand, the evidence is not sufficient to entirely exclude the heart as a factor in the production of cardiac failure.

In this conflict of theories it has been suggested that there is possibly a third factor aside from the heart and the vasomotor center, though of necessity more or less intimately associated with them, which controls the flow of blood.

Henderson[3] found that he could produce shock by overventilating the lungs, and he believed that shock may be produced by removing the CO_2 from the blood and tissues (acapnia) as a result of excessive respiration. He has also shown that, although in the early stage of shock there is a diminished cardiac output, the blood-pressure is maintained by a constriction of the peripheral arterioles. He stated further that stasis of blood in the veins causes an abolition of venous tone. He maintained also that shock supervened in laparotomies on account of the escape of carbon dioxid from the exposed peritoneum and intestines.

Widal,[4] in a recent publication, maintains that the numerous and widely diverse clinical manifestations of shock are traceable to a single mechanism, namely, the process of destruction of colloids (colloidoclasis) or, in other words, an upset of the physical balance between colloidal structures.

Bainbridge and Parkinson,[5] in their experiments on rabbits and dogs, showed that the sudden opening of any body cavity was sufficient in some cases to produce shock, causing a reduction of general sensibility and a relaxation of the musculature. Continued handling of the contents of the body cavity, in a subject of low resistance, leads to more or less complete shock. The cardiovascular or respiratory systems were predominantly affected.

It is well known that extensive incisions or burns of the body surface may produce collapse or shock, and that violent mechanical agitation of some sensitive part without actual destruction of tissue may bring about the same condition.

It has been shown by Dale and Laidlaw[6] that the injection of histamin in small quantity into experimental animals produces symptoms similar to those which occur in traumatic and surgical

shock. The outstanding symptom is deficiency in the volume of blood, due in part to a passing of the plasma out of the blood-vessels and to the retardation of the peripheral blood flow. It is thought that this oligemia is due to loss of tone of the capillaries and that the minute vessels become more permeable and allow the escape of plasma through their walls.

Histamin is not the only substance which is capable of producing a shock anaphylaxis. Other substances which seem to bear some relation to histamin, such as bee poisons and certain bacterial products, are capable of producing effects similar to that of histamin. The outstanding toxic result of all these substances consists of poisoning of the capillary endothelium. A similar result is produced by certain metallic poisons such as arsenic and the gold salts. Any of these poisons if introduced into the general circulation produce a shock-like prostration.

Shock occasionally follows the intravenous administration of salvarsan. Before this product was accurately standardized shock was not an uncommon reaction. In these cases the shock is supposed to be due to the poisoning of the endothelial cells of the capillaries. When meningococcus serum was first introduced shock occasionally followed its intraspinal administration.

Bayliss and Cannon[7] maintained that an important factor in the production of shock consists of the absorption into the general circulation of certain products which are liberated when tissues are injured. They showed that shock might be produced by injuring a limb in which the nervous connections have been severed from the rest of the body. If the blood-vessels were securely clamped they found that no shock occurred. They concluded that by preventing the entrance of toxic products into the blood shock was avoided.

Pearce and Eisenberg[8] have suggested that anaphylactic shock and intoxication are characterized by a condition of low blood-pressure very similar to that seen in other varieties of shock and collapse, and that they are produced without trauma or other conditions which are usually considered in the etiology of surgical shock.

The animals which went into anaphylactic shock, as well as those who suffered from intoxication, showed a fall of blood-pressure to 20 to 30 mm. of mercury. This tended to persist in anaphylactic shock, but relatively rapid recovery occurred in peptone intoxication. In both conditions there was an extreme congestion of the large venous trunks of the splanchnic area. At the same time there was observed an intense anemia of the centers of the medulla oblongata.

As a result of their experiments Pearce and Eisenberg believe that the low pulse pressure produced by the injection of toxic products is due primarily to the small amount of blood passing through the heart. They claim that changes in the heart are secondary and not primary. Respiration is not altered except in so far as it is affected by the anemia of the medullary centers.

Mautner[9] studied anaphylactic shock and the effect of the injection of histamin and peptone. He claimed that these injections produced a constriction of the efferent blood-vessels of the liver, and that this occlusion of the venous blood-stream led, on the one hand, to a stasis of the vessels of the intestines, and, on the other hand, to a deficiency in the blood to the heart, resulting in a fall of blood-pressure in the systemic circulation. If this disturbance in the distribution of the blood occurs it would be important to know what relation such a change in the vascular flow plays in the production of collapse.

The classical experiments of Romberg[10] and his pupils have shown that collapse in the course of infectious diseases is not primarily cardiac, but of vascular origin. They think that a paralysis of the vasomotor center plays the important part. In some of the cases, however, a primary constriction of the blood-vessels of the liver occurs which in the case of infectious diseases is caused by the effect of retrograde metabolic products. A fall in blood-pressure results. In the case of the food intoxications of infants it has been assumed that the intoxication is due to an overwhelming of the organism by large molecules of protein. This ingested protein has been recovered as such from the urine. It has also been demonstrated that the intestinal wall of infants suffering from severe alimentary disturbance is more

permeable than in the normal infant. In this connection Moro has shown that in intoxication the conditions for poisoning with protein products are ideal because a large amount of amins may be derived from the protein of milk.

Lust[11] studied the action of large molecules of colloids of egg-albumen and determined that these molecules may pass through the intestinal wall and find their way into the circulation, and Hirsch and Moro[12] fed peptones and peptid substances and succeeded in producing a condition similar to alimentary intoxication.

Circulatory failure in infectious disease may be explained primarily by paralysis of the vasomotor centers, and it is doubtful if the heart participates at the onset. Romberg observed that in pneumococcus infections the heart does more work during the period of the infection than under normal conditions, and that although there may be some vascular paralysis and a threatened collapse, nevertheless the heart remains compensated for a time.

This is true not only in pneumococcus infections but also in grippe and diphtheria. These investigations also show that collapse occurring during an infection does not depend upon direct damage to the heart, but is rather the result of vessel paralysis.

Animals who had suffered from cardiac weakness brought about by diphtheria toxin were artificially asphyxiated. The asphyxiation caused heart symptoms indicating a degree of myocardial degeneration. Animals who had previously suffered from pneumococcus infection did not show similar cardiac symptoms. The heart tended to recover itself in the animals who had suffered from pneumococcic infections if they were allowed to breathe normally, that is, if the process of asphyxiation was stopped. If the heart has been affected by diphtheria toxins it is more difficult to resuscitate the animal, demonstrating that in pneumococcic infection the heart is scarcely involved, while in diphtheria infection the cardiac muscle is seriously affected. The diphtheria toxin probably damages the neuromuscular apparatus of the organ.

Certain accessory factors to the production of shock may be mentioned, such as muscular relaxation, decrease of intra-abdominal pressure, and impaired respiration. Each of these tends to decrease the amount of blood returned to the heart.

Some investigators have laid stress upon the degenerative changes of the cells of the central nervous system. Such changes, however, are the cause and not the result of shock. Dolley states that identical changes in the nerve-cells are produced by hemorrhage and shock.

In Crile's experiments in the production of artificial cerebral anemia he showed that a certain proportion of nerve-cells were permanently lost and a great many were damaged. In addition, there was some nerve-fiber degeneration. He thinks that the changes which occur in cerebral anemia are in certain respects identical with shock.

In collapse conditions occurring in infantile alimentary intoxication we find a desiccation of all the tissues of the body. The blood undergoes a drying-out process as well, and this diminution of blood volume leads to a decrease in the rate of circulation. The amount of blood which passes through a given portion of the body is thereby diminished and, as a result, the capillaries and arterioles in the peripheral areas become constricted. This may be shown by comparative blood-counts made in the capillaries and the veins. By this very simple technic it may be demonstrated that when there is constriction of the arterioles, the contained hemoglobin and red cells are increased, whereas the number of red cells and hemoglobin in the veins are diminished.

The increase of red cells and hemoglobin in the arterioles and their diminution in the veins indicates stasis in the capillary bed. This gives rise to a lowered blood-pressure, a serious alteration in the mechanism of the circulation, and is one of the conditions which indicate circulatory failure and shock.

This disproportion occurs in patients who have suffered from shock, and the greater this difference, the more profound the shock. When this stagnation continues, the outcome is unfavorable because circulatory failure and cardiac weakness en-

sues, while if the congestion diminishes, improvement is taking place.

Wallace considered the subject from another angle. Besides shock from loss of blood, loss of heat and toxemia, he recognized a kind of shock without external wounds and with very slight internal injuries. The distinguishing feature was a concentration of blood which, if marked, caused death in spite of all treatment. This variety should be classified as the pure or nervous shock. In such cases rest and warmth might be followed by an elevation of blood-pressure and operative interference usually caused fatal collapse. In this group the pulse was slow and the termination nearly always fatal, the condition of the patient suggesting an injury to the upper part of the spinal cord.

SYMPTOMATOLOGY

The symptoms of collapse are so well known that only a brief statement of the outstanding features need be noted: Alterations in the respiratory and cardiac actions; modified mental state; loss of power of voluntary and involuntary muscles; diminution of secretion of urine; and in all cases except in acute infections there is lowering of the body temperature. The pulse frequently becomes accelerated and is easily compressible.

The face is usually blanched, though sometimes the superficial veins contain a considerable amount of blood. It presents an anxious appearance, and when coma or unconsciousness has occurred it may be expressionless or mask-like. It is frequently shrunken and pinched; the chin droops; the nose is pointed and the tip is cold; in extreme cases the eyes have lost their lustre. The patient, as a rule, is extremely weak; the eyes are rolled upward; the lips are pale or cyanotic.

The skin is pale gray and appears bloodless. A cold perspiration occurs on the surface of the body. Respirations, as a rule, are rapid and frequently sighing. Breathing is labored and the Cheyne-Stokes variety may occur. Air-hunger is not infrequent, but is more indicative of hemorrhage than of shock.

The muscles are relaxed. In extreme cases the patient

maintains the position in which he is put. Very often voluntary muscular power is lost. The function of digestion is diminished and vomiting and relaxation of the sphincter of the bowel sometimes occur. He is more or less unconscious, though he may be delirious, showing considerable mental excitation and continuous rambling speech. Frequently he is confused mentally, talks irrationally and incoherently.

In babies who are suffering from alimentary intoxication the temperature may be elevated or subnormal. The pulse becomes almost imperceptible and the blood-pressure is diminished. The fontanel becomes markedly depressed and very soon one notices dark rings about the lower eyelids. The face seems to shrink and become smaller. The color of the skin becomes ashen gray and more or less livid. The skin becomes inelastic and loses its turgor. The extremities become cool. The interior of the mouth and the exhaled breath seem cooler than normal. The respirations at times are rapid. The urine may show the presence of acetone and diacetic acid and, in some cases, a small amount of albumin and casts are present. More or less unconsciousness or actual coma may be present.

The symptoms in these infants are not uniform and the severity of the collapse is variable. Moderately severe cases may recover under proper treatment, but the extreme cases of collapse usually terminate fatally.

TREATMENT

Patients who show symptoms of shock or collapse should be placed in bed as soon as possible. External heat should be applied by the use of hot blankets or hot-water bottles. Hot drinks should be given if the patient is not nauseated or vomiting. In the case of infants weak tea may be administered.

The cardiac stimulants are time-honored remedies, although the opinion at the present time is not unanimous as to the value of these drugs.

Camphor.—The injection of camphor, in the form of camphor oil, has been and still is in general use. Pharmacologic experiments show that the results obtained give no constant ef-

fects. Laboratory reports and careful clinical studies seem to indicate that camphor fails as a stimulant in shock and circulatory failure. Clinicians of experience, on the contrary, will still maintain that camphor given subcutaneously or intramuscularly or even by mouth is of value in collapse conditions.

In the light of more recent investigations concerning shock and collapse there should be no hesitancy in saying that the administration of camphor used as a cardiac stimulant is not calculated to relieve the diminished flow or volume of blood, the capillary stasis, or the paralysis of medullary centers. Even if it succeeds as a cardiac stimulant, it does not act to relieve the other and more important changes which have gone on in the organism. Therefore it should not be considered a potent or important factor in the treatment of shock and collapse.

Epinephrin is fugitive in its action and does not produce a sustained effect on the circulation. When given hypodermically it raises blood-pressure by acting on the muscles of the vessel wall or upon the small nerve filaments supplying these vessels. It acts as a neuromuscular stimulant. The injection of adrenalin produces blanching of the skin, and it is generally believed that its effect causes constriction of the peripheral vessels. Thus it may be assumed that adrenalin acts as a tonic stimulus to the vessels and in this way enables the blood to circulate freely through the vascular bed, thus relieving stasis. The blood-pressure within the arteries and the deep veins is raised, and while the drug constricts the vessels of the skin, it permits the blood to pass into the muscles.

Adrenalin also acts upon the heart first by accelerating it and then slowing it. Slowing of the heart occurs when the blood-pressure is raised as a result of vagal stimulation. Epinephrin is, therefore, indicated when there is evidence of an acute circulatory collapse with rapid pulse and falling blood-pressure. The drug may be given intravenously and may be considered of special value in the infectious diseases, particularly in pneumonia. On account of its transitory effect it should be repeated at short intervals.

Cahn and Steiner[13] observed that while some individuals

react to subcutaneous injection of adrenalin by a strong rise of blood-pressure, in others this reaction is almost or entirely absent. In certain individuals a transient glycosuria may occur after adrenalin.

Schiff and Balint[14] recommend that atropin be given hypodermically in small doses before epinephrin is administered. They consider this advisable in those individuals who react unfavorably to epinephrin by exhibiting chills, tremors, and fainting attacks. During life the adrenal glands suffer from an insufficient secretion as a result of injury, and in many of the acute infections, such as diphtheria, pneumonia, and influenza. Such patients should be given the suprarenal gland extract, but not in large doses. With excessive doses a fall of pressure may occur, resulting in death. Epinephrin is preferably given hypodermically. It may be given in small doses on the tongue and allowed to be slowly absorbed. In the stomach it undergoes some chemical change which diminishes its blood-pressure-raising effect.

Pituitary Extract.—The action of pituitary extract is similar to that of epinephrin, the difference being rather a matter of intensity. Pituitrin acts less rapidly, but its effect is said to be more prolonged. In one instance its use may be preferred to that of epinephrin, that is, where there is considerable tympanites or abdominal distention with cardiac embarrassment. Pituitrin stimulates the atonic intestinal muscles and the heart is relieved of mechanical interference produced by the meteorism.

Caffein.—It has been shown that caffein may cause a rise in the total blood flow without a corresponding rise in pulse-rate. When the flow of blood to the right heart is inadequate, caffein enhances the circulatory rate by increasing the venous supply. It raises the blood-pressure by its stimulating effect on the vasomotor center. It slows and steadies the heart by stimulating the vagus center. It acts not only on the heart itself but also has been shown to cause vasodilation on account of a peripheral depression of the vasoconstrictor mechanism. There is every evidence to believe that caffein is a useful remedy in the treatment of circulatory failure.

Digitalis.—There is no universal agreement as to the value of digitalis in the treatment of circulatory failure during acute febrile conditions, though recent electrocardiographic studies show that digitalis exerts the same effect on the heart in febrile conditions that it does in the non-febrile state.

If during the course of an infectious disease the pulse becomes small and soft and the extremities cold and blue, the indication is to aid the circulatory organs. Digitalis is usually selected for this purpose. The use of digitalis in pneumonia has not been found beneficial in most instances. On the other hand, if the heart is defective in one way or another before the onset of pneumonia, digitalis may be indicated. There is no basis for giving digitalis in every case of pneumonia. We are not surprised to find that digitalis has no effect in the acute infectious diseases because we have learned from Romberg and his students that the collapse in the acute infectious diseases is not due to a cardiac insufficiency, but is caused by a paralysis of the smaller vessels. It is for this reason that the drugs which act upon the vasomotor center are more useful—caffein, adrenalin, pituitrin, and atropin.

There are still some who believe that digitalis is effective in the circulatory failure of typhoid, and the drug continues to be used in the acute infectious diseases, after hemorrhages, prolonged anesthesia, and in collapse in general. One should not expect much from the use of digitalis in these cases, and the vasomotor stimulants are more definitely indicated.

Treatment with Saline Solutions.—In former times saline infusion administered in one way or another was generally used in the treatment of shock and collapse with the idea of restoring the fluid part of the blood in the artery. This method frequently failed, and Bayliss[15] endeavored to explain why a relatively large number of patients did not respond to this treatment by saying that the saline solution escaped rapidly from the blood-vessels. Consequently, Bayliss added 6 per cent. gum arabic to the solution, and it was soon observed that this infusion maintained the raised blood-pressure longer than the saline solution without gum. The use of this solution was empiric.

Rogers, in his studies of cholera patients, found that if they were given gum solution intravenously their condition was aggravated, with the development of dyspnea and cyanosis. Rogers thinks that the toxins of cholera may be retained in the blood when gum solution is administered.

Other investigators have maintained that the infusion of saline solution by rectum and the taking of fluids by mouth are the best and most rational methods of restoring the volume of blood. It has been thought that the rectal saline infusion stimulates bone-marrow more than does the transfusion of blood, saline, or gum solution given intravenously.

Erlanger and Gasser[16] also recognized the increased concentration of the blood and observed that the blood-corpuscles tended to stagnate in the venules and capillaries. These authors thought that the capillaries dilated and that plasma exuded through their walls. Nutrition of the tissues suffered by these changes. On this basis they added glucose to the saline solution with the idea that it nourished the tissues. The injection of 5 to 10 per cent. glucose solution has come into general use and surgeons and internists alike report excellent results from its use. Moore[17] argues against the use of infusions of sodium bicarbonate solutions by pointing out that the acids of the blood are increased in shock, but considers that there is some increase of the alkalinity of the blood.

Owing to respiratory conditions, the patient consumes more oxygen and loses more carbon dioxid than in health. This may be due to the fact that less air is inhaled. The carbon dioxid may be formed in smaller quantity, though the amount which escapes need not be diminished; indeed, it may be retained in the body and because of its presence the blood is rendered more alkaline. Thus Moore thinks that "alkalosis" is a term which designates the condition more accurately than the term "acidosis." In view of this condition of alkalosis Moore insists that the administration of bicarbonate of soda is not indicated and may do harm.

The advantage of the rectal infusion of saline enemata is that considerable quantities of fluid can be introduced without

inflicting pain. Large quantities of fluid are absorbed in this way.

Subcutaneous infusion offers a satisfactory method of introducing fluids into the body, although it is a method not unattended by pain. Too large quantities should not be given in one area. If the fluid is excessive in quantity, great pressure is brought upon the superficial tissues and sloughing may result.

Adrenalin chlorid solution, 15 minims, 1 : 1000, may be added to 150 to 200 c.c. of normal salt administered subcutaneously. This should be given slowly and has the advantage of introducing fluid into the circulation and at the same time obtaining the extended action of adrenalin. A continuous infusion of 1 : 20,000 adrenalin solution administered at the rate of 2 c.c. per minute is a desirable method.

Intraperitoneal Method.—Physiologic salt solution, or Ringer's solution, or 5 to 10 per cent. glucose in 2 or 3 per cent. sodium bicarbonate solution may be given by the intraperitoneal route. The use of glucose intraperitoneally has been abandoned by us on account of its irritating effect. Adrenalin, 10 or 15 minims, may be added to the saline solution which is to be used for intraperitoneal injections. This method may be employed in place of enteroclysis, hypodermoclysis, or intravenous injection. Intraperitoneal injection may be employed; 100 to 250 c.c. may be given to small infants, and in larger infants and young children 300 to 400 c.c. may be introduced without causing abdominal distention or embarrassed respiration. The fluid is readily and rapidly absorbed, and if caution is exercised there is practically no danger of infection or injury to the bowel.

The injection may be repeated once in twelve or twenty-four hours. Indeed, in young infants who have suffered from collapse as a result of alimentary and nutritional disturbances, this plan of treatment has given most satisfactory results and may be considered a life-saving procedure. The use of a sharp-pointed needle should be avoided. A trocar, such as is used in spinal punctures, is preferable.

I have observed that after the administration of saline

intraperitoneally the blood-pressure will be increased 10 to 12 mm., and will remain so for several hours after the injection.

Transfusion of whole or citrated blood may be required in the treatment of collapse of infants and young children. It is particularly indicated in very anemic patients. If the hemoglobin has fallen to 25 per cent. or less, blood transfusion is urgently required.

Blood should not be introduced rapidly because of the possibility of acute dilatation of the heart in a patient with a weakened myocardium. Blood may be given through the longitudinal sinus in young infants and, in older children, any accessible vein may be selected. The external jugular is frequently the vessel of choice.

The value of transfusion in infections is not only due to the fact that it supplies an amount of blood which is lost and relieves anemia, but it also introduces new defense bodies and possibly increases the patient's immunity to infection.

The amount of fluid injected intravenously varies from 75 to 150 c.c., depending upon the infant's age and weight. This holds true for saline infusions as well as blood transfusions.

Mustard.—The favorable effect of the external application of mustard in circulatory failure of infants and young children has long been recognized. An excellent method for its employment consists in soaking a baby blanket or bath sheet in a gallon of hot water to which has been added a handful of mustard. The blanket is wrung out and the baby enveloped within it. The mustard pack may be changed once or twice during a half-hour period. If the skin has reacted and becomes red, the baby's body should be well washed with warm water and rubbed with vaselin. The baby should then be allowed to rest.

This is a valuable therapeutic measure for the heart failure which occurs in pneumonia and nutritional disturbances, or in any condition in which heart failure is threatening or impending. The pulse-rate is diminished, the systolic blood-pressure increased, and, in general, a successful circulatory stimulation results.

Inhalation of Carbon Dioxid.—The inhalation of carbon

dioxid mixed with oxygen has been suggested in the treatment of cardiac collapse in accordance with the views of Yandell Henderson. This method requires further clinical corroboration before its efficiency can be considered proved.

Rest and **sleep** are important factors in the treatment of collapse and shock. Excessive manipulation should be avoided. Operative interference should be deferred and excessive treatment should be dispensed with.

The surroundings should be kept quiet. Anxious friends should be enjoined from disturbing the rest and quiet of the patient. Sleep should be induced. Sometimes a moderate dose of paregoric in infants or an appropriate dose of codein or morphin in an older child will do more to relieve shock and collapse than all other medication which may be employed. Indeed, it may be a life-saving measure.

If it is found necessary to adminster a narcotic drug, the use of fluids should be continued, using one of the methods to which we have already referred.

The last word has not been said on this important theme. Since the most authoritative opinion is not unanimous on the nature of shock and collapse, there can be no unanimity in the treatment.

Notwithstanding the more or less unsettled state of our knowledge concerning the nature of shock, the one outstanding symptom, which is an unfailing expression of circulatory failure, is a lowered blood-pressure, and this is almost invariably present.

When life is ebbing and it hangs by a slender thread, we are frequently tempted to employ heroic and energetic measures. The lessons recently learned from the studies of circulatory and nervous behavior in shock and collapse teach that many of the older therapeutic measures were unfounded and futile.

The so-called cardiac stimulants have a narrow range of usefulness. Rest and quiet, restoration of the normal circulation by raising the lowered blood-pressure and the introduction of fluids into the body, are the most important factors in the treatment of shock and collapse.

BIBLIOGRAPHY

1. Crile, George W.: Keen's Surgery, 1914, vol. i, Chap. xxii, W. B. Saunders Co. An Experimental Research into Surgical Shock, Philadelphia, 1899. Crile, George W., and Lower, E. W.: Surgical Shock, W. B. Saunders Co., Philadelphia, 1921.
2. Howell, W. H.: Contributions to Medical Research, dedicated to Victor Vaughan, 1903.
3. Henderson, Y.: Acapnia and Shock, Amer. Jour. Phys., xxi, 126, 1908; xlvi, 533, 1918.
4. Widal, F., Abrami, P., and Brissaud, E.: Clinical Shock, Presse Med. xxviii, 181, 1920 (abst. Jour. Amer. Med. Assoc., lxxiv, 1609, 1920).
5. Bainbridge and Parkinson: Lancet, i, 1269, 1907.
6. Dale, H. H., and Laidlow, P.: Histamine Shock, Jour. Phys., lii, 355, 1919.
7. Cannon, W. B.: The Course of Events in Secondary Wound Shock, Jour. Amer. Med. Assoc., lxxiii, 174, 1919.
8. Pearce, R. M., and Eisenberg, A. B.: A Study of Low Blood-pressure, Arch. Int. Med., vi, 218, 1910.
9. Mautner, H.: Die Wirkung der Shockgifte in ihrer Beziehung zur Klinik, Mon. f. Kind., xv, 283, 1919.
10. Romberg, E., Passler, V., and Rolly: Experimentelle Untersuchungen überkreislaufstörungen, Deutsch. Arch. f. klin. med., lxiv, 652, 1899; lxxvii, 96, 1903.
11. Lust, F.: Zur Nutzanwendung der Lehre von der Bedeutung des Eiweisses für die genesender alimentären Intoxikation, Münch. Med. Wchnschr., lxviii, 1353, 1921.
12. Hirsch, A., and Moro, E.: Weitere Untersuchungen über alimentäres, Fieber. Jahr. f. Kind., lxxxviii, 314, 1918.
13. Cahn, R., and Steiner, B.: Absorption of Epinephrin, Jahr. f. Kind., xcix, 44, 1922.
14. Schiff, E., and Balint, A.: Über den Einfluss des Atropins auf die blutdrucksteigerndewirkung des Adrenalins bei Kindern, xciv, 1, 1921.
15. Bayliss: General Discussion on Shock, Proc. Royal Soc. Med., xii, 1918–19.
16. Erlanger, J., and Gasser, H.: Action of Hypertonic Gum Acacia and Glucose After Hemorrhage, Amer. Jour. Phys., l, 149, 1919.
17. Moore, B.: The Increase of Alkalinity of the Blood in Shock, Lancet, cxcvii, 473, 1919.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

DUODENAL ULCER COMBINED WITH CHOLECYSTITIS

Two patients who have recently entered Mercy Hospital are examples of a combination which is not unusual in clinical work, namely, gall-bladder disease in association with ulcer of the duodenum.

Case I.—The first case is that of a young woman, twenty-nine years of age, single, who entered the hospital January 29, 1923 with the following history:

She complained of pain in the epigastrium which had lasted for about four or five years; of recent vomiting, belching, constipation; and of a comparatively small loss of weight. When she was a child she had chickenpox, measles, and scarlet fever. Aside from these disturbances she was comparatively well until 1917, when her present trouble began. As is usual in cases of this type, a diagnosis of appendicitis was made, and in 1917 her appendix removed. She states that following its removal she had an attack of what was diagnosticated as "ether rheumatism." I am ignornat of such a kind of rheumatism. To me rheumatism means a generalized infection of the joints from some focus of infection, and when a patient tells me that following an operation for appendicitis rheumatism developed I am not disposed to ascribe it to ether, but rather to some infection associated with the appendicitis or an active focus not discovered at the time of the operation.

As to her present history, the patient states that at first the attacks of trouble were spaced a few months apart, that they lasted three or four weeks at a time, but that as the years have gone by the intervals between the attacks have gradually grown

less extensive, so that now she is continuously sick. Her weight loss has not been very great. At the time of the operation for appendicitis she weighed about 109 pounds, and in the interval between then and now she has lost 12 pounds, so that she now weighs about 97 pounds. Her chief complaint is *pain*. She described it as located in the epigastrium, as not radiating to the thorax, in character rather sharp, coming on about half an hour after she eats. The duration of the pain depends upon the amount and kind of food eaten. Fats, meats, and pastry increase the pain. This fact is of slight bearing as a possible indication of gall-bladder infection. The pain is relieved by drinking hot water or by taking bicarbonate of soda.

The patient is also troubled with gas and belching which come on immediately after she has eaten. At first there is a feeling of distress in the epigastrium and she states that something seems to be pressing against the heart. This sensation of upward pressure does not disappear until the patient ceases belching, which does not occur until about two or three hours after she has eaten. The larger the meal, the longer the duration of belching. (This sensation of gas and belching is usually due to hyperacidity.) The patient also complains of vomiting. This happens two or three times a week and occurs usually about one-half hour after eating. So far as she has been able to observe the only thing which influences the vomiting is the amount which she eats. If she eats very sparingly there is no vomiting. If she eats more than the usual amount vomiting seems to follow. Constipation is also complained of and is rather pronounced. She has some signs of general infection. To be sure, there are no inflamed joints and no complaints of "neuritis," but she is bothered not infrequently with headache, and occasionally with a bad taste in her mouth and a coated tongue.

On analyzing these symptoms one is immediately struck with certain contradictions. The periodicity of the seizures is rather suggestive of ulcer of the duodenum rather than of the stomach, and the comparatively slight loss of weight is consistent with duodenal ulcer rather than gastric ulcer. The gas and belching are of slight differential value diagnostically be-

cause they merely indicate a probable hyperacidity which is common both to chronic appendicitis, gastric and duodenal ulcer, cholecystitis, and cholelithiasis. The pain which she complains of is indicative rather of duodenal ulcer than of anything else. It is located in the epigastrium and is confined there. As a rule, pain due to biliary tract infection radiates to the thorax and is found between the shoulder-blades, in the right shoulder, and also in the right pleura and mediastinum. Nevertheless there is one point in regard to the epigastric distress which is worth noting, and that is the complaint of cardiac disturbance which she ascribes to upward pressure of gas. As a matter of fact there is no such thing as upward pressure of gas in these cases with a secondary mechanical effect upon the heart. We see this in innumerable fluoroscopic examinations where the stomach gas bubble is of the usual size with no upward pressure upon the diaphragm at all. As a rule such cardiac symptomatology is indicative of associated cholecystitis rather than of ulcer, either of stomach or duodenum. In pure cases of duodenal ulcer cardiac symptomatology is never complained of. In cases of ulcer associated with cholecystitis, however, it is not infrequent for patients to make complaint of cardiac symptoms, and particularly of seizures taking place in the night.

The vomiting which she complains of is not a symptom of pure cases of uncomplicated duodenal ulcer. Whenever vomiting is found in a patient complaining of symptomatology suggestive of ulcer of the duodenum a complication should always be sought. Ordinarily in biliary tract infection the patient vomits not once but several times, the early vomiting containing food and the later vomiting containing chiefly bile and mucus. This patient has vomited, as a rule, only once, occasionally twice after meals, but she has shown bile in the contents very frequently while under our observation. Whenever bile appears in the vomitus the condition should always be attributed to associated disease, either on the part of the gall-bladder or of the associated peritoneum or of the appendix. One would conclude, therefore, from a consideration of the symptom of vomiting which has been rather prominent among her specific com-

plaints that she is suffering not alone from a probable duodenal ulcer, but that she has also some associated biliary tract pathology.

The constipation that she complains of is suggestive of ulcer rather than of an infected gall-bladder. As a rule, cases of uncomplicated cholecystitis and cholelithiasis are not troubled with constipation, whereas universally cases of duodenal and gastric ulcer suffer from that condition. If there be a combination of ulcer with gall-bladder symptomatology, however, the ulcer will determine the presence of constipation.

Most important in all of her complaints of specific symptomatology are a few signs of infection. She complains of occasional headaches, of coated tongue, and of a bad taste in her mouth. Occasional headaches may occur in any individual whether suffering from ulcer or not. They might, for example, be due to eye strain or sinus trouble; but if on looking over a patient no obvious explanation for such headaches can be found, it is a fair conclusion that they are due to other causes. If such headaches occur in an individual complaining of signs of ulcer of the duodenum in association with coated tongue and a bad taste in the mouth, one is surely justified in concluding that there is some focus of infection in the body, and the obvious location of such a focus in such a patient is the biliary tract.

With these preliminary considerations in mind the patient was given a Rehfuss fractional test-meal in order to determine the acidity. The Rehfuss meal consists of 8 ounces of tea with $\frac{1}{2}$ ounce of lactose. The Rehfuss tube is passed within a half-hour, and eight samples are collected at half-hour intervals, each one of which is examined for total acid, free hydrochloric acid, mucus, bile, blood, and sugar. It was found in the case of this patient that she had a double acid curve, that is to say, the acidity at the second half-hour period reached the first maximum, falling to low in the fourth half-hour period, reaching a second maximum in the sixth half-hour period, and falling slightly in the eighth half-hour period. The curve of combined acid was paralleled exactly by the curve of the free hydrochloric acid which also showed two crests. At the end of four

hours' time the total acid was 32 and the free hydrochloric acid 20. In a normal stomach the acidity at the end of four hours should be 0 as to free hydrochloric acid and but a few points above in the case of the combined acids. It was also noted in the examination that a good deal of blood was present in each of the specimens, reaching its maximum in the sixth and falling toward the eighth. This may have been traumatic blood. It is not safe to pay too much attention to blood which is obtained in the fractional test-meal because ordinarily the irritation of the tube itself is sufficient to cause a certain amount of bleeding.

Repeated examinations of the feces over quite a period of time were subsequently made for the purpose of noting occult blood. She was under medical attention from January 29th to March 8th, and during all of this time her stools were never quite free from blood by either the Weber or benzidin test. A sample of vomitus which was examined on February 23d contained blood. On the same day the feces showed 4+ blood by the benzidin test. It is unnecessary to enumerate all the examinations of feces which were made, but it is enough to say that out of ten examinations of the feces between the time of entrance and the time of operation, nine contained blood, only one being free. This constant presence of blood in the feces not due to bleeding of the gums or to other possible ingestion of blood is strongly presumptive of a bleeding point somewhere in the alimentary canal.

On February 1st, two days after the patient entered the hospital and the day following the Rehfuss test, the patient was taken to the *x*-ray room and examined fluoroscopically. This examination showed the barium emulsion passing through the esophagus without obstruction and freely entering the stomach. The latter was of the atonic type, the greater curvature reaching about 3 inches below the crest of the ilium in the median line, so that it is evident that the patient has quite a degree of gastroptosis. (Parenthetically it may be noted that gastroptosis is not an infrequent sign of mild pyloric block. One used to look upon Glenard's disease as always congenital in origin. We used to say that patients with very low stomachs

were born that way. As a matter of fact, we find a large number of patients in whom the stomach reaches a very low level without associated floating kidney, either right or left. Here the falling of the stomach is undoubtedly due to some interference with the outflow of food through the pylorus.)

Gastric peristalsis was quite active, apparently as a compensatory measure to overcome the low position of the stomach. The pylorus closed off squarely and no filling defect was observed in the greater curvature even up to the very beginning of the pylorus. On the lesser curvature, however, in the pyloric antrum and close up against the pylorus a persistent defect appeared. Beyond this the duodenal cap formed fairly normally, except that it showed a great deal of irregularity at the point where the duodenum turns to the right. This inequality of filling was slightly suggestive of a possible adhesion between stomach and adjacent structures, preventing the proper and even filling of the whole of the duodenal cap. The rest of the fluoroscopic examination was quite normal.

A series of plates was then taken which showed quite clearly the filling defect on the lesser curvature just at the pylorus with a little tendency for a displacement of the cap to the left, so that the alignment of the cap and pyloric antrum is a little distorted. We looked upon this defect, which shows quite clearly in one of the plates, as due to scar tissue formation consequent upon a prepyloric ulcer. The pylorus itself is not blockaded because there is no five-hour retention in the plate taken at the end of five hours' time. There was no evidence of pylorospasm in her history. That the ulcer is active seems likely from the continued presence of blood in the feces.

Because of the results of the Rehfuss test-meal and fluoroscopic examination and an analysis of the symptomatology we concluded that there was present a prepyloric ulcer which was active and which was the cause of the continuous epigastric pain, but that probably there was also some associated gallbladder disease, this latter conclusion being based upon the somewhat slender evidence of the vomiting of bile and of the associated signs of infection, chiefly headaches, coated tongue,

and bad taste. Coated tongue and bad taste in the mouth are not found in the hyperacidity of gastric and duodenal ulcer. On the contrary, in these cases the tongue is almost universally clean and red. It is only when there is associated biliary tract infection that the mouth begins to get foul.

The patient accordingly was put upon the regulation treatment for ulcer. Her diet was at first only milk, and it was pushed very vigorously. During the first week she received 4 ounces of milk every hour with frequent alkaline powders and sufficient oxid of magnesia to take care of her constipation. The second week, there being some improvement, the amount of milk was changed and each feeding was doubled, as was also the interval of time between the successive doses, so that she got 8 ounces of milk every two hours. She was bothered, however, a good deal in the second week by evident pylorospasm, which we attempted to relieve by tincture of belladonna, but with indifferent success. We persisted in the treatment for a period of one month, until February 28th, at which time we determined that because of the continuation of the gas and belching, pain and occasional vomiting, that she would have to be operated upon. It was determined beforehand that a gastrojejunostomy would probably have to be done and possibly the gallbladder removed. We had no means of telling whether stones were or were not present because the patient had never been jaundiced and had never had an attack of biliary colic. Our plates showed no stones.

On March 2d she was taken to the operating-room at 8.15 A. M. Dr. John F. Golden opened the abdomen by a large paramedian incision from the ensiform process to the umbilicus. The stomach was delivered and revealed an indurated area about the size of a 5-cent piece, evidently due to a chronic peptic ulcer situated on the prepyloric portion of the lesser curvature exactly at the place where it was supposed to be. A posterior gastrojejunostomy was performed in the usual way. On incising the mesocolon adhesions were found between the posterior gastric peritoneum and the mesocolon, and these had to be liberated. They were evidently due to an associated peritonitis following the ulcer.

After the gastrojejunostomy portion of the operation was finished the gall-bladder was explored. It was found to be quite dilated and somewhat thickened and to contain a very large number of small stones. The patient subsequently had the pleasure of counting them, and I think there were 179. The gall-bladder was removed in the usual way and the common duct explored and found free from stones. All the raw surfaces were covered with peritoneum and a rubber drain was inserted and the abdomen closed with a drain.

Following the operation the patient had at first a slight rise of temperature, which quickly fell, and in due course of time she has made a splendid recovery. The operation was on March 2d; the patient is still in the hospital (April 25th) apologizing at the present time for her great appetite and the amount that she can eat. She has gained all of the 12 pounds which she had lost and a little besides. Following the operation, as is usual in all our gastrojejunostomies, close attention was paid to the presence of acetone. On March 4th acetone was present in two specimens sent to the laboratory; present 1+ in two specimens on the 5th and 2+ in one specimen on the 5th. Late in the evening of the 5th acetone was reduced to a trace and by the 6th it had disappeared. We combat this acetonemia by bicarbonate of soda and glucose. Postoperatively it is my custom always to order four 500 c.c. rectal injections daily of a 5 per cent. glucose and 2 per cent. bicarbonate of soda solution at six-hour intervals. These are given by the drop method at the rate of from 40 to 48 drops per minute. Given in this way at six-hour intervals there is always a resting period of about three hours between the injections, so that they do not become burdensome to the patient.

On the second day after the operation it is our custom to give sips of hot water by mouth, and on the third day we start with fruit juices which contain fructose, a variety of dextrose which does not have to be digested, but is absorbed as such into the blood-stream. We usually give loganberry juice or apple cider, both of which are, as a rule, well tolerated by the patients. Some brands of apple cider are very sour and cannot be used.

We try to get sweet apple cider containing little malic acid and a good deal of fruit sugar. Adulterated cider containing cane-sugar is worse than useless because cane-sugar has to be digested before it can be absorbed, and these patients are not capable of digesting levulose. We lessen on the second and third days the amount given by proctoclysis and increase the amount given by mouth. Usually on the third day as much as 500 c.c. are given by mouth, so that only 3 pints are given by proctoclysis at eight-hour intervals, and ordinarily about the fourth or fifth day all proctoclysis is stopped. At times it happens on the fifth day that it is possible to feed these patients a good deal of soft food without harming them.

This procedure has been followed in the case of the present patient, and she has made a splendid recovery. All pain, vomiting, gas, and belching have entirely disappeared. Had the patient not had a complete diagnosis with a complete operation we should not have had a cure. The associated gall-bladder pathology undoubtedly had quite a large part in the production of the symptomatology. I am quite certain that our belief that vomiting is always indicative when found in the presence of duodenal ulcer of associated pathology elsewhere is exemplified by this case and that such vomiting is a valuable differential symptom, as is also the evidence of infection, even though, as in this patient, comparatively slight.

Case II.—The second patient was one year older than the preceding patient, but she had a similar history. She complains of the same periodicity that the preceding patient complained of, but she has not been sick for so long a time, her illness dating back to about one year. The loss of weight was greater, it being 20 pounds in the last three months. She complains of pain in the epigastrium and in the region of the stomach, not radiating, and associated with considerable soreness. This pain is not definitely related to food. It is almost always present and usually is intensified about fifteen minutes after she eats, and, unlike the pain of duodenal ulcer, it is not relieved by alkalies or by food. Like the preceding patient, she also vomits.

She states that since last December she has had an almost continuous disagreeable taste in her mouth in the morning and that she occasionally vomits bile. She is constipated, but she has no signs of infection and no headaches, nothing except the coated tongue and the disagreeable taste in her mouth.

Without going into details in the differential diagnosis of her case it is enough to say that she was examined in the same manner as the preceding patient, but that she showed a different Rehfuss curve. The first specimen which was removed showed a total acidity of 42 and free hydrochloric acid of 30, but in the third specimen the acidity had fallen considerably, and kept on falling, so that it reached its lowest point in the eighth specimen. There was no double curve as in the preceding case. It was, however, quite noteworthy that instead of recovering, as is usual in most stomach cases, comparatively small amounts measured in cubic centimeters, we recovered from the stomach large amounts; for example, at the end of the first half-hour 6 ounces were recovered, although she ingested but 8, and at the end of the second half-hour period 3 more ounces were recovered, with 5½ ounces at the end of the three-and-a-half-hour period; so altogether in the eight samples or four hours there was collected from the stomach 21 ounces of fluid, although the ingestion was but 8.

When she was fluoroscoped she showed the emulsion passing freely into the stomach, which was somewhat lowered in position, the greater curvature reaching about 2 inches below the crest of the ilium. No filling defect was found, however, in the stomach itself. The duodenum, on the other hand, showed a marked contraction at the junction of the second and third portions with what might be construed to be evidence of adhesions between duodenum and gall-bladder. On the lesser curvature and on the greater curvature just in front of the antrum no defects were found, but the plates show a narrow band of bismuth connecting the duodenal cap with the antrum. The pathology here is evidently confined to the duodenum. The five-hour plate shows what one might expect to find—a considerable degree of five-hour retention. When one compares

the large amount of fluid recovered at the time of giving the Rehfuss meal with this five-hour retention one is forced to the conclusion that there is some obstruction which is responsible for both the retention of barium and the retention of fluids. Because of the degree of five-hour retention it was not felt advisable to delay operation.

She entered the hospital on March 6th, was examined on the 7th and 8th, and operated on the 9th. The case was recognized as being in many respects similar to the previous one, which was operated upon on March 2d, so we rather counted on finding ulcer of the duodenum with gall-bladder pathology. Again we could not determine the presence or absence of stones because of the lack of pain and jaundice. We could only conjecture the possibility of stones being present because of the resemblance to the previous case. The operation was also done by Dr. John F. Golden and the same median abdominal incision made from the ensiform process to the umbilicus. Exploration of the stomach revealed an indurated area with some adhesions of the omentum about the lesser curvature and over the pylorus. These adhesions were due to an associated peritonitis consequent upon an old ulcer in the first portion of the duodenum. A gastrojejunostomy was done in the usual way. After that operation had been finished the gall-bladder was explored and was found adherent to the bowel and omentum. The wall was thickened and the gall-bladder was filled with large numbers of stones ranging in size from a very large one, almost the size of a hickory nut, in the neck of the gall-bladder, to very small ones about the size of fine gravel. No stones were palpated in the ducts. The gall-bladder was removed without opening in the usual way. There was some hemorrhage from the liver attachment, but this was controlled by suture. Two rubber drains were inserted and the abdomen was closed.

This patient was treated in precisely the same manner as the preceding one. She showed following operation quite a little vomiting for the first few hours, with some acetone findings. She was given the same kind of saline injections of glucose and bicarbonate of soda followed by sips of water, broth, logan-

3163

berry juice, orangeade, and cider. She made a recovery even more rapid than the preceding patient and left the hospital April 13th entirely well in every respect and on a rather full diet, consisting of cereal, coffee, toast, boiled eggs, chicken broth, custard, jelly, and similar things. Instructions were given her gradually to enlarge the diet and to report for further advice from time to time as needed.

These 2 cases are shown for the purpose of emphasizing the fact that not infrequently an ulcer of the duodenum or prepylorus, even in the comparatively young, unmarried people of twenty-nine and thirty years of age, may be complicated by rather severe gall-bladder pathology. We are accustomed in general to regard gall-bladder pathology as belonging in the forties, and we are correct in looking upon the maximum incidence as occurring at that time of life, but we must not forget that many cases of chronic appendicitis and ulcer of the stomach and duodenum are associated with secondary lymphatic infection of the biliary tract. Sometimes this infection reaches very grave proportions. In each of these cases there was the scar of an ulcer; in each of these cases a gastrojejunostomy was required for the cure of the patient. Had the operation stopped with the doing of the posterior no-loop gastrojejunostomy there would have been only an imperfect cure. The only value of the operation to these 2 patients lies wholly in the fact that all of the abdominal pathology was taken care of and not part of it. Too much emphasis cannot be placed upon the careful interpretation of abdominal symptomatology. It is never enough to make a diagnosis of some abdominal condition. One must not be content until a complete diagnosis of all the abdominal conditions has been made. If one gets to looking upon cholecystitis and cholelithiasis as associated end-products of ulcer of the stomach and duodenum, one will always be on the alert for these complications.

CLINIC OF DRS. CHARLES A. ELLIOTT AND WALTER H. NADLER

WESLEY MEMORIAL HOSPITAL, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

CHRONIC GALL-BLADDER DISEASE

IT is the object of this clinic to present what appears to be a working hypothesis of the physiology of the gall-bladder in the light of experimental work, and to correlate with this hypothesis the well-known clinical manifestations of chronic gall-bladder disease. The case histories of a number of patients with chronic cholecystitis will be presented and discussed. The far-reaching effects of chronic gall-bladder disease on the general economy and the importance of early recognition and treatment will be pointed out.

The internist is constantly confronted with the difficulty of detecting chronic gall-bladder disease, often the underlying cause of many obscure gastro-intestinal manifestations. The symptoms of chronic cholecystitis are apt to be so obscure, indefinite, or atypic that a certain diagnosis may not be made. Experimental work on the physiology and pathology of the gall-bladder is, therefore, scrutinized with keen interest in the hope of obtaining exact criteria which may be applied in clinical medicine.

Physiologic Considerations.—The conception of the physiology of the gall-bladder has been greatly modified by the results of recent investigations. It is evident that the gall-bladder in man has a definite and important function. With the accumulation of data on the behavior of the gall-bladder under experimental conditions the theory that it is a vestigial organ must be definitely discarded.

Mechanically, the gall-bladder acts in conjunction with the sphincter of Oddi in controlling the storage of bile and its passage into the duodenum. Regarding the passage of bile into the duodenum the sphincter of Oddi is the determining factor, whereas forceful contractions of the gall-bladder seem of less importance. The musculature of the gall-bladder which contracts rhythmically five to eight times a minute (Bainbridge and Dale), a general characteristic of smooth muscle, together with the well-developed elastic fibers of its wall, maintains a positive pressure in the bile passage. Between meals slight intermittent relaxations of the sphincter of Oddi permit the escape of small amounts of bile and maintain a constantly intermittent flow through the gall-ducts. With the passage of food into the duodenum the sphincter relaxes more completely, most of the bile being discharged at this time. The sphincter of Oddi, retaining bile in the bile passages, is able, in experimental animals, to resist a pressure of approximately 700 mm. of water (Oddi). The gall-bladder, under experimental conditions, exerts a maximum pressure at the height of contractions of only approximately 220 mm. of water (Freese).

As a diverticular reservoir the most important function of the gall-bladder seems to be its ability to concentrate bile by absorption of watery elements through the mucosa and lymphatics, reducing the bulk of the bile six to ten volumes. In this regard the mucosa of the gall-bladder acts differently from that of the gall-ducts. The former abstracts material from the bile, thus concentrating it; the latter secretes a clear liquid ("white bile") which is added to the bulk of the bile, diluting it (Reus and McMaster). The total amount of bile secreted by the liver in twenty-four hours probably equals the amount of urine secreted by the kidneys during the same period. If 1500 c.c. of hepatic bile is secreted in twenty-four hours this amount is reduced by absorption to 150 or 200 c.c. of cystic bile. The 40 c.c. content of the average gall-bladder, therefore, represents roughly 250 to 400 c.c. of hepatic bile.

After removal of the gall-bladder the bile passages dilate (Judd and Mann). The evident causes of this dilatation are the

obstruction produced by the contractions of the sphincter of Oddi sufficiently powerful to resist a pressure of 700 mm. of water, and the increased volume of hepatic bile unconcentrated for lack of functionating gall-bladder mucosa.

The reciprocal innervation theory of the sphincter of Oddi and the gall-bladder (Meltzer), of which much has been written of late, has opened up a profitable field for clinical investigation. This theory seems to explain many of the manifestations of gall-bladder physiology. While not definitely proved, it may well be retained as a working basis. The diversity of opinion as to the value of non-surgical "duodenal drainage of the gall-bladder" as proposed by Lyon may be explained by the presence of such variable factors as differences in amount of gastric and duodenal content, uncertain position of the duodenal bucket, regurgitation of bile into the stomach, etc. Although apparently based on sound physiologic principles, the Lyon test has proved of little practical value in diagnosis because of difficulty in interpreting results.

In conditions associated with an increase in the blood of certain constituents normally excreted by the liver a physical predisposition toward the production of gall-stones occurs because of the concentration of these constituents of the hepatic bile in the gall-bladder, and because of the intermittent stasis of the thickened bile. Thus, the increased cholesterin content of the blood in pregnancy and in continued fevers, and the increase of bile (blood) pigments in the blood of patients with hemolytic icterus present conditions favorable for the deposit of cholesterin and pigment stones. This theory of the formation of gall-stones seems more rational than one which hypothesizes a chronic infection of the contents of the gall-bladder itself.

Infection of the gall-bladder occurs by way of the blood-stream, usually through the portal system, the wall of the gall-bladder being infected before its contents. Indeed, Graham found it difficult or impossible to produce cholecystitis in animals by infecting the contents of the gall-bladder unless the cystic duct was at the same time ligated. The presence of chronic hepatitis in practically all cases of chronic cholecystitis (Graham)

indicates the importance of the spread of infection from the gall-bladder to the liver by way of the lymphatics, and emphasizes the value of the early recognition and treatment of gall-bladder infection.

Clinical Manifestations.—In the diagnosis of chronic cholecystitis, as of all gastro-intestinal diseases, the greatest importance should be placed on the clinical history. The greater the experience one has had in the diagnosis of obscure abdominal lesions, the more one appreciates the value of a complete history. The proper interpretation of the so-called "irritative gastro-intestinal symptoms" provoked by any extragastric abdominal lesion presents a field for profitable clinical investigation. It is evident that any pathologic condition in the abdomen may provoke abnormal physiologic processes, and, consequently, symptoms frequently attributable to the gastro-intestinal tract. The patient usually ascribes such symptoms to the stomach. Certain combinations of these symptoms are more or less distinctive of the different extragastric lesions, among them of gall-bladder pathology. Symptoms of chronic gall-bladder disease are apt to be described by the patient as indigestion, biliousness, flatulence, acid stomach, or chronic constipation. They may be masked by less obviously related complications of a chronic cholecystitis, as chronic hepatitis, chronic myocarditis, coronary sclerosis, arthritis, or recurring urticaria. The stomach, small intestine, appendix, colon, and kidney may be under suspicion. The true nature of such symptoms may not be clear before the advent of a typical attack of gall-bladder colic or jaundice.

A history of previous pregnancies, continued fevers, such as typhoid, sedentary life, or, obesity has long been considered of such importance that one hesitated to make a diagnosis of cholecystitis without at least one of these predisposing factors having been present. The modern conception of the physiology of the gall-bladder supports the rationale of this view.

The symptoms of gall-bladder disease may be divided into two groups—the irritative gastro-intestinal manifestations, which are only suggestive, and the colic and jaundice which are characteristic. Such typical symptoms as the latter are so well

known that they will be referred to but briefly at this time. It is desired rather to describe the irritative features which have been of aid in the diagnosis, especially of the more obscure cases.

Many of the *irritative gastro-intestinal symptoms* of cholecystitis occur in other abdominal conditions, but certain of them seem fairly characteristic of gall-bladder disease. Among the most common of these symptoms are a sense of fulness, tension, weight, or pressure in the epigastrium which comes on immediately after eating; a sense of satiety after taking but a few mouthfuls of food or drink; gas, belching, and flatulence especially soon after eating; and a sense of sudden and uncontrollable nausea occasionally associated with faintness or salivation. The patient invariably ascribes these symptoms to the stomach. They are aggravated markedly after certain foods, especially meat and greasy foods, and after a full meal. These symptoms are not so important in themselves as in their association with the general clinical picture and in their persistence. They may last for years, occurring in more or less indefinite attacks which recur at irregular intervals. Such disturbances are apt to have started after a continued fever or during the child-bearing period, rarely in young people. They are not relieved by catharsis, differing thereby from the irritative gastro-intestinal symptoms of chronic appendicitis. They differ from the "indigestion" of duodenal ulcer in that attacks are less definite and of shorter duration, and are of greater irregularity and more frequent occurrence. Vomiting when produced as an irritative symptom of gall-bladder disease is apt to be persistent, a retching which continues all night without relief, as compared to the slight or rare vomiting in chronic appendicitis, and the immediate relief by vomiting in peptic ulcer. The exact significance of the above irritative symptoms may not be apparent until the onset of such late manifestations as gall-stone colic or jaundice.

On physical examination the findings are variable. Usually they are scant and of questionable aid in diagnosis. The following, when present, are of more value: a complexion usually muddy, often with a suggestion of jaundice; tenderness at the

right rib edge, especially on deep inspiration; a tender spot in the back to the right of the vertebral column; relative contraction and resistance of the upper portion of the right rectus muscle; palpable and tender liver edge or gall-bladder. There may be slight elevation of temperature and a moderate leukocytosis. In cases with localized inflammation about the gall-bladder, with adhesions to the duodenum, stomach, or colon, the clinical picture is usually complicated by fever, leukocytosis, pain on eating, moving, and vomiting, together with symptoms referable to the adherent organ. Pain referred to the right shoulder is a significant symptom in such cases. Recurrent urticaria, interpreted as a probable allergenic manifestation, has occurred in several of our cases. Chronic myocarditis and chronic arthritis in certain patients who presented many of the above irritative symptoms have been interpreted as complications of chronic gall-bladder infection.

Gall-bladder colic presents certain fairly characteristic features. Colic which occurs suddenly and without a history of associated gastro-intestinal (irritative) symptoms is probably caused by a pure cholesterin or a pigment stone. All other types of gall-bladder colic which may occur in any grade of severity are associated with a history of gastro-intestinal disturbances and are due to cholecystitis. The gall-bladder attack is largely mechanical in nature. It has a definite relation to meals in that it is apt to occur within a few hours after a large meal, such as a banquet. It occurs typically in the middle of the night several hours after the patient has gone to bed. The typical pain is epigastric, more on the right than on the left side, intermittent, "colicky." It radiates in any direction—to the back, the right shoulder, or the precordium. Pain of sufficient intensity to warrant calling a physician in the middle of the night to administer a hypodermic of morphin which may need to be repeated before relief is obtained is suggestive of gall-bladder colic. Significant also is the fact that the attack is apt to be sudden in onset, "bursting in type," or "sharp, as though the body were transfixed with a knife." The patient is restless and rolls with agony. He vomits frequently without relief. There

is slight, if any, temperature, leukocytosis, or jaundice. The abdomen has no board-like rigidity, but may be palpated in spite of marked rigidity of the upper portion of the right rectus muscle as compared with the left side. The attack rarely lasts more than a few hours. Localized tenderness in the right upper abdomen persists and the liver may be somewhat swollen and tender for a day or two. Such attacks recur usually at rather long and irregular intervals, especially after overeating. Alkalies, quality of food, and cathartics have no effect in preventing attacks.

Chemical analysis of the stomach contents has been of little aid in the diagnosis of gall-bladder disease. There are, however, certain rather significant features in the behavior of the stomach. By fractional examinations in a large series of demonstrated gall-bladder cases it has been found that the hydrochloric acid curve rises rapidly, reaching a maximum acidity at the time of emptying of the stomach, which is early, usually within an hour and a half after the test-meal. This curve, fairly characteristic of a large number of cases, may be distinguished from the average curve of other typical abdominal lesions. It is interpreted as evidence of an irritable hypertonic stomach which empties itself rapidly of a highly acid content. In another group of long-standing gall-bladder disease with stones there is an absence of free hydrochloric acid.

The *Roentgen findings* in chronic gall-bladder disease have, in our experience, except for the evidence of adhesions and the rare demonstration of outlines of stones, been chiefly of negative value in eliminating pathologic lesions of the gastro-intestinal tract. Cholesterin and pigment stones are rarely of sufficient density to be demonstrated on radiograms. Stones of more than the average density, usually impregnated with inorganic salts, may be demonstrated, but these constitute only a small percentage. Negative shadows, due to the lesser density of the cholesterin stones as compared with the surrounding tissues, are more frequently seen, but furnish suggestive rather than positive evidence of stones. The outline of the gall-bladder itself is but rarely seen. Adhesions from the gall-bladder to the duodenum,

pylorus, or hepatic flexure of the colon, distorting these structures, are more frequently seen and are of distinct value in diagnosis. A high-standing and transverse stomach, apparently showing increased tonus, is frequently seen.

CASE REPORTS

Case I.—Mr. Le V. (103,198); age forty-six years; admitted to Wesley Memorial Hospital January 2, 1923. History of four short attacks of epigastric pain, the first occurring in July, 1921. He was awakened from a sound sleep by a sharp pain localized about the navel; it lasted for two hours, when it was relieved by a drink of whisky. Attacks had lasted for one and a half to three hours. Nausea had been associated, but no vomiting, fever, or jaundice. In the intervals between attacks he felt quite well. The last and most severe attack began at 3 P. M. on the day before admission to the hospital. An hour after the noon meal he experienced cramp-like pain in the region described, increasing in severity until it became almost knife-like in character. The pain remained localized for seven hours. The following day there remained only a dull ache in the gall-bladder region and in the epigastrium. No serious previous illness. Physical examination showed a plethoric, well-nourished man, not acutely ill, no jaundice. Oral infection present. Slight, rather diffuse tenderness in right hypochondrium and epigastrium. Liver edge palpable, hard, not tender, 3 fingerbreadths below the costal margin. On January 7th he was awake all night with pain in the right upper quadrant. There was definite muscle resistance; a tender mass, moving with respiration, was palpable in the gall-bladder region. Blood Wassermann negative. White blood-cells 11,400. Fasting stomach contained 18 c.c. of material (free HCl 0, total acid 28); thirty minutes after test-meal acidity of 0 and 80, one hour after meal 44 and 60; stomach empty after one and a half hours. Roentgen examination of the gastro-intestinal tract revealed no pathology; radiograms of the gall-bladder region were negative. Gall-bladder drainage (Lyon's method) showed three fractions; no pus-cells, but many bacteria of all kinds.

Laparotomy January 11, 1923 (Dr. M. T. Goldstine). A chronically inflamed gall-bladder containing stones and a long, adherent, injected, retrocecal appendix were found. Cholecystectomy and appendectomy were performed. Microscopically great thickening of the gall-bladder wall together with cellular infiltration, some edema, and small areas of hemorrhage were noted.

Diagnosis.—Chronic cholecystitis and cholelithiasis; recent recurrent cholecystitis; infection about teeth.

Discussion.—Definite, though relatively slight, attacks of gall-bladder colic had recurred during a period of eighteen months. No history could be obtained of previous symptoms referable to the appendix or gall-bladder, although it is probable that they were present and disregarded. Microscopic examination of the gall-bladder showed that the last attack was associated with definite acute inflammation despite apparently normal temperature and only slight leukocytosis. The acidity curve as determined by fractional examination of the stomach contents is of the type frequently found in gall-bladder disease.

Case II.—Dr. W. (103,483), aged fifty years, admitted to Wesley Memorial Hospital on January 17, 1923. History of distress in epigastric region and of nausea and belching after eating. He had lost 10 pounds in the previous two weeks. Attacks of distress were first noticed about November 1, 1922. During November and December he had from six to eight recurrences, none of them severe. Within ten to fifteen minutes after a meal distress appeared in the right upper abdomen associated with belching of gas; this lasted one and a half to two hours or longer, and then gradually disappeared. There was only slight soreness on deep palpation. The effect of soda was never tried. After January 1, 1923 symptoms became more severe and frequent, appearing practically after every meal until two days before admission to the hospital, when a milk-and-toast diet gave relief. Appetite was at no time impaired; no vomiting. At the age of fourteen years the patient was jaundiced for several weeks; at twelve years he had diphtheria, followed by acute

nephritis, and at forty-five years diphtheria followed by multiple neuritis which did not entirely clear up for six months. At seventeen years he had rheumatic fever which kept him in bed for two to three months and was said to have caused a mitral lesion. At forty-two he had a mild arthritis, but kept at work. He has had four to five slight attacks of pain over the appendix region at intervals of about eight months. Examination showed a rather stout plethoric man of fifty years; no jaundice. Epigastric tenderness just below the xiphoid cartilage. Heart normal. White blood-cells 9200. Blood-pressure and urine normal. Blood Wassermann negative. Fasting stomach contained 10 c.c. of material (free HCl 0, total acid 15); maximum total acidity 32 at time of emptying (one and a half hours), no free HCl at any time. Roentgen examination of the gastrointestinal tract was negative except for a suspicion of appendical involvement; no evidence of gall-stones in radiograms.

Gall-bladder pathology was suspected, but not strongly enough to advise operation. About a month after leaving the hospital the patient suffered an unmistakable attack of gallstone colic; at operation (Dr. H. M. Richter) chronic gall-bladder inflammation with stones was found.

Discussion.—This case illustrates the difficulty in making a definite diagnosis in the absence of a history of colic. Yet, the history suggested gall-bladder disease. A more careful history and further observation should have made the diagnosis evident. This patient had no free hydrochloric acid in stomach contents.

Case III.—Mrs. A. (99,642), aged sixty-three years, admitted June 22, 1922. History of repeated attacks of pain in the right upper quadrant and in the epigastrium radiating toward the right scapula, and of loss of weight and strength. She had suffered some twenty-five to thirty attacks in the past eight years. More recently, especially since a sore throat and influenza in November, 1921, attacks have been more severe and frequent, occurring at weekly intervals. The early attacks consisted of sudden pain in the right upper abdomen without associated nausea or vomiting, relieved by morphin. Some

attacks, though severe, were of short duration and did not require a hypodermic. In more recent attacks vomiting of green material came on early and might continue every hour for days. Tonsillitis has occurred at least every winter for many years. The patient has 3 children. Twenty years ago a pelvic operation was performed. No typhoid fever.

Examination showed a well-nourished woman with considerable tenderness in epigastrium and in right upper quadrant, where a mass seemed to be palpable. Liver and spleen not palpable. No jaundice. Blood-pressure 164/86. Urine normal. White blood-cells 9700. Blood Wassermann negative. Fasting content of stomach 25 c.c. (free HCl 24, total acid 60); maximum acidity one and a half hours after test-meal (74 and 104); stomach empty after two hours (70 and 82). Roentgen examination of the gastro-intestinal tract was negative except for a large ovoid shadow, as of gall-bladder or of right kidney, around which the hepatic flexure passed; this shadow contained many irregular shadows.

Laparotomy on June 28, 1922 (Dr. A. B. Kanavel), showed a large, edematous gall-bladder distended with fluid and containing several stones. There was no pathology about the hepatic flexure. Appendix was small and of normal appearance. Cholecystectomy was performed. Recovery was prompt.

Diagnosis.—Chronic cholecystitis and cholelithiasis.

Discussion.—A history of repeated attacks of colic during a period of eight years, during which time, if a diagnosis was made, operation had never been advised. There is a possible relation between attacks of sore throat and exacerbations of gall-bladder inflammation.

Case IV.—Mrs. B. (104,422), aged fifty-six years, was referred for general examination March 13, 1923 because of extreme restlessness, shortness of breath on exertion associated with momentary localized lumbar pain, and with pain localized beneath the ensiform cartilage in an area the size of a dollar, and prolapse of the uterus present for fifteen years. There had been 6 normal pregnancies. The perineum was badly lacerated

twenty-eight years ago. At thirty-one years of age the patient was ill for a month with rheumatism. Menses are still regular at the age of fifty-six years. Many years ago she had several attacks thought to have been appendicitis. At first no gastro-intestinal symptoms of any kind were admitted, but careful inquiry revealed that she had occasional attacks of "abdominal gas" as well as stomach disturbances which seemed to be due to dietary errors and which were promptly relieved when vomiting was induced. The substernal and lumbar pain appearing with shortness of breath only after exertion was not associated with palpitation. These symptoms were, nevertheless, considered suggestive of cardiac distress. The patient was short and well nourished. There was left-sided cardiac hypertrophy; no murmurs. Abdomen negative. Infection present about the teeth. Cystocele, rectocele, and prolapse of uterus. Blood-pressure 150/75. Blood Wassermann negative. Urine normal. White blood-cells 5850.

The operation advised because of the pelvic pathology was performed on April 9th by Dr. Kanavel. Supracervical hysterectomy was performed and the adherent left tube, the cystic left ovary, and the chronically inflamed obliterated appendix were removed. The gall-bladder was palpated and found to contain many stones.

Discussion.—The apparently latent character of many cases of chronic cholecystitis and appendicitis is here illustrated. A vague history of previous appendicitis and of slight, almost disregarded, irritative stomach symptoms was obtained only with effort. These were scarcely sufficient to warrant a diagnosis of the pathology of the gall-bladder and appendix, which was found at operation.

Case V.—Mr. W. H. M. (103,373), aged sixty-five years, presented himself for examination about a year ago. A diagnosis of chronic gall-bladder infection with stones was made from a history of typical attacks of pain together with vague symptoms of indigestion which had been present at times for many years. He had, moreover, been jaundiced for four or five days in one attack. Roentgen examination was negative.

The patient would not consent to an operation. He remained well until January 8, 1923, when, about an hour after a banquet, he became nauseated and developed severe epigastric pain which radiated to the back and shoulders. The next day he began to hiccup and the abdomen became distended. Nausea continued. No fever, chills, or vomiting. On January 10th he was admitted to the hospital. He was pale, cyanotic, dyspneic, and appeared to be in a condition of collapse. Pain was present in the gall-bladder region; it was aggravated by the frequent singultus. The heart area was broad (right sternum to 1 inch beyond midclavicular line); localized systolic murmur at apex; rate rapid; rhythm irregular, tumultuous. The abdomen was greatly distended and hard, especially on the right side. The liver seemed to extend to the level of the navel; this area was tender and rigid. Several ounces of "coffee-ground" material were obtained by aspiration of the stomach. Acute cardiac decompensation with acute dilatation of the stomach occurring during a severe attack of gall-stone colic was suspected.

He spat up coffee-ground material until January 16th; on this date the scleræ were icteric; a little food was retained for the first time. Singultus was less frequent and distressing at this time, but continued at intervals until January 22d. Exacerbations of pain and tenderness over the gall-bladder region occurred on January 17th and 26th, but no definite involuntary muscle rigidity could be determined. On February 1st an electrocardiogram showed normal mechanism. General condition was much improved.

On February 6th a laparotomy was performed by Dr. H. M. Richter. A thickened gall-bladder containing several stones; stone in the cystic duct; enlarged hard pancreas; many areas of fat necrosis in omentum (verified by microscopic examination); and a few adhesions about the appendix were found. Cholecystectomy was performed. Recovery was uneventful. At the present time the patient is able to work in his office a few hours each day. He feels well except for pain in the right shoulder anteriorly, associated with hyperesthesia, present for the last five years. Glycosuria has never been demonstrated.

Diagnosis.—Chronic cholecystitis and cholelithiasis; chronic pancreatitis with recent acute attack.

Discussion.—This patient had had indigestion for years, culminating about two years before operation in typical attacks of gall-stone colic. On at least one occasion there was temporary obstruction of the common duct. Surgical interference was refused. Interstitial changes gradually occurred in the pancreas and in the liver. The patient ate lightly and remained well for a year, when overeating appeared to bring on an attack which predisposed to or was associated with a recurrent cholecystitis. Acute pancreatitis followed, and for a time it was feared that he would not survive the accompanying shock. This complication together with the chronic interstitial changes in the pancreas and probably in other organs might have been avoided by early operation.

Case VI.—Mrs. E. (95,040), aged sixty-five years, admitted to the hospital October 4, 1921. Complaint: "Indigestion," attacks of pain in right upper quadrant of abdomen; urticaria, and swelling, diagnosed angioneurotic edema. Indigestion, especially heartburn, relieved by soda, had been present for years. In years past she has had mild attacks of nausea and vomiting at intervals of about six months. Two years before admission to the hospital the left cheek became swollen and red, subsiding in twenty-four hours. Thereafter at frequent irregular intervals similar localized swellings involving various parts of the body appeared; sometimes there were none for months. On one occasion when the throat was affected symptoms were alarming. At times typical urticarial lesions were present in place of or associated with the swellings. In March, 1921 occurred the first severe attack of abdominal pain; the next day swellings appeared on various parts of the body. Quincke's disease was diagnosed. In July, 1921 there was a second attack of severe pain in the right upper quadrant of the abdomen; swellings followed. September 26th she had a third attack, beginning as indigestion, and followed by excruciating pain in the right upper abdomen. The acute pain lasted forty-

eight hours. For a week there was great discomfort, abdominal distention, fever, and chilliness. Stools were clay colored for two days; no definite jaundice. Giant urticaria followed. No history of typhoid fever. One child is living and well.

Examination showed a stout woman of sixty-five years, uncomfortable because of abdominal distress and because of a red, tender swelling above the right eye. A tender mass of the shape of the gall-bladder was palpable in the right hypochondrium; there was extreme tenderness and rigidity in this region. Blood-pressure and urine normal. Blood Wassermann negative. White blood-cells 15,350. Temperature normal. Protein sensitization tests negative. Gall-bladder drainage (Lyon's method) yielded only about 100 c.c. of material, none of which was of the dark color characteristic of the second or gall-bladder fraction. Fractional test-meal: fasting content 10 c.c., free HCl 8, total acid 22; maximum acidity one and three-quarter hours after test-meal, free HCl 72, total acid 92. Roentgen examination showed normal esophagus, stomach, and duodenal cap; duodenal cap angulated to the right; point of tenderness not directly over the cap, but in close relation to it; normal emptying time of stomach. Appendix retrocecal and apparently adherent. Radiograms of the gall-bladder region showed no evidence of stone.

At operation on October 7, 1921 (Dr. A. B. Kanavel) an enlarged, very greatly thickened gall-bladder, the wall of which was in some areas hard as cartilage, densely adherent to the pylorus and duodenum, and containing a single stone the size of a large walnut, was found. The appendix was chronically inflamed. The gall-bladder and the appendix were removed. Following the operation the patient remained free from symptoms until the summer of 1922, when giant urticarial lesions reappeared after a "cold." They were present also for several weeks during the past winter.

Diagnosis.—Chronic cholecystitis and cholelithiasis; chronic appendicitis; urticaria apparently due to sensitization from gall-bladder infection.

Discussion.—Gall-bladder inflammation with irritative symp-

toms ascribed to the stomach and, for the most part, disregarded, had evidently been present for many years. Predisposition to the formation of a gall-stone may have been furnished by pregnancy and subsequent obesity. Recurrent gall-bladder infections seem to be responsible for the urticarial lesions appearing in the two years prior to operation. About seven months before operation typical attacks of colic and infection (fever, chilliness, urticaria) appeared. These were ascribed by some physicians to "internal hives." Before the appearance of the severe attacks a definite diagnosis would have been difficult. The urticarial lesions overshadowed the other symptoms. In this case the Lyon test was of value, though its interpretation was, at the time, not clear.

Case VII.—Mrs. A. E. G. (103,869), aged sixty-nine years; admitted to Wesley Memorial Hospital on February 8, 1923. History of "sore spot" in right abdomen for twenty-five years; of attacks of "cramp" in epigastrium characterized by severe pain, vomiting, chill and fever, for over two years; and of gradual loss of weight in past three to four years. At least twenty-five years ago she had a very severe attack of pain in the right lower abdomen; vomiting, fever, constipation, and loss of consciousness, diagnosed by various consultants as appendicitis, floating kidney, etc. For six weeks hot and cold packs were applied alternately. Ever since there has been a dragging sensation in the right lower abdomen. Four years ago she had a sudden attack of pain and vomiting diagnosed as appendicitis. Since then she has been miserable, with soreness on the right side, aggravated by exercise and relieved at times by soda. Two years ago a severe attack of epigastric pain, vomiting, and fever was diagnosed gastritis. In July, 1922 she had stomach distress and vomiting after all food. Always she was conscious of her right side, with, at intervals, acute pain in the upper abdomen. For example, in December she had acute pain; also twice during one week in January. On January 25th an attack was aborted by soda and by induced vomiting, but the next day a cramping pain referred to the back and followed by chill and fever appeared. On January 31st morphin was given for

the relief of severe sharp pain and tenderness in the epigastrium. On February 4th and 5th she had repeated similar attacks, culminating in sharp, severe pain which lasted for over twelve hours. On February 6th she had fever; vomiting was induced, with some relief. On February 7th jaundice and pruritus were present; stools were white and urine contained bile. The patient was poorly nourished. Heart area was broad; systolic blow at apex. Blood-pressure 162/90. Tenderness and considerable resistance in right upper quadrant of abdomen; liver and spleen not palpable. Blood Wassermann negative. White blood-cells 9700. Fasting stomach empty; maximum acidity one and one-quarter hours after test-meal, free HCl 80, total acid 100; stomach empty after one and three-quarter hours, acidity 60 and 90. Roentgen examination of the gastro-intestinal tract made prior to the appearance of jaundice was negative except for slight tenderness in the right lower quadrant, where a shadow thought to represent the appendix was made out, yet failed to show definite evidence of pathology. Radiograms of the gall-bladder region were negative.

Laparotomy, February 13, 1923 (Dr. A. B. Kanavel). Gall-bladder small, atrophic, bound to liver and bowel by numerous adhesions; dilated common duct containing a single stone the size of a marble; chronic obliterative appendicitis. The gall-bladder, common duct stone, and appendix were removed. Drainage from the gall-bladder site continued for one month.

Diagnosis.—Chronic appendicitis; chronic cholecystitis, and cholelithiasis; common duct stone.

Discussion.—This patient gave a definite history of acute appendicitis twenty-five years ago followed by the irritative symptoms of chronic appendicitis. Gall-bladder pathology, evidently present for at least four years, altered the clinical picture. For the most part symptoms were "irritative" and due, at least in part, to adhesions about the gall-bladder. The symptoms complained of were gastric. Definite, though relatively slight, attacks of gall-bladder colic appeared, and finally, about six weeks before operation, there were symptoms of intermittent obstruction of the common duct.

Case VIII.—Miss H., (97,874), aged fifty-six years, was admitted to Wesley Memorial Hospital on March 15, 1922 complaining of chills and fever, intermittent jaundice, "indigestion," and loss of weight (18 pounds in a year).

From June, 1919 to June, 1920 the patient was treated for cystitis. In June, 1920, after the fourth of a series of vaccine inoculations, she developed a chill and severe pain ascribed to gall-stones. Before a cholecystectomy was performed at the end of the same month she had suffered repeated attacks. At operation gall-stones were found and the gall-bladder was said to be full of pus. About a year later (July, 1921), following a "cold," she developed pain in the back beneath the right shoulder-blade and down the right side of the abdomen. Jaundice, never present before the operation, was noticed at the same time. Thereafter she was jaundiced off and on; stools were often clay colored; pruritus marked. Chills and fever began in December, 1921 and recurred perhaps twice in five days, or might be absent for as long as sixteen days. There is no history of typhoid fever; thirty years ago "blood-poisoning" following a carbuncle on the neck kept her in bed for several months.

The patient was a tall, pale, flabby woman, definitely jaundiced. The abdomen, which showed the scar of operation in the gall-bladder region, was soft and pliable; liver and spleen not palpable; no tenderness or rigidity. The blood-serum contained bile. Wassermann test negative. No increased fragility of red blood-cells. Red blood-cells 4,030,000; white blood-cells 13,600; Hgb. 70 per cent. Stools, which were pale and contained only traces of bile, were free from blood. Blood-pressure and urine normal. Fasting content of stomach measured 7 c.c. (no free HCl, total acid 6); stomach empty after one and one-quarter hours, total acidity 12, no free HCl at any time. Roentgen examination showed slight hesitation at the cardia and a slight, but inconstant irregularity of the pylorus and cap; no shadows as of gall-stones. A diagnosis of common duct stone was made.

Laparotomy was performed on March 29, 1922 by Dr. Kanavel. The liver was found to be small and pale, with many adhesions to the diaphragm and about the site of previous

operation. Pylorus not obstructed; stomach normal. Pancreas normal. Some adhesions about the spleen, which was not enlarged. Appendix appeared normal. Twelve large soft stones together with mucoid plugs were removed from the distal portion of the common duct. Recovery was uneventful and the patient has been quite well in the past year.

Diagnosis.—Common duct stones with intermittent obstruction in a patient whose gall-bladder had been removed.

Discussion.—This case illustrates typical common duct obstruction and biliary infection. From the size and number of stones present nineteen months following a cholecystectomy performed by a competent surgeon it must be assumed that they were formed in the interval following operation. Infection present at the time of the first operation when the gall-bladder was said to contain pus, or possibly caused by the "cold" which antedated the later symptoms, together with dilatation of the common duct and bile stasis, predisposed to the formation of stones.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

ACUTE LYMPHATIC LEUKEMIA (SMALL-CELLED TYPE)

THE 3 cases which I wish to present to you today I have selected because they are all fairly closely allied and all belong to the group of the so-called blood diseases. A second and perhaps more important reason is because of the difficulty in diagnosis and because we have been able to establish these with a reasonable degree of certainty.

The patient before you is a laborer, a native of the United States, fifty-two years of age. He came to the hospital this morning in a rather stupid frame of mind and complaining principally of shortness of breath, which seemed to be quite extreme, cough, pain in the right side of the chest, and in the throat.

Onset and Course.—He states that about a week ago he began to cough, and this was continued in about the same way, never being extreme. He raises very little sputum and, in particular, never has had any evidence of hemoptysis. The pain in the right side has been present for a somewhat longer time. He has only noticed it off and on, and ten days or two weeks ago it became more continuous and has really been bad enough to bother him very much for only about a week. The shortness of breath came on a week ago, before which time he says he could do anything that the other men working with him could do. It has, however, increased at a very rapid rate, and at the present time he can hardly walk without complaining of very marked dyspnea. He states that he has never had any chills and does not know whether he has had any fever, but thinks he has not. He has lost no weight so far as he knows. His

strength, he thinks, was as good as ever until just the last day or two, but now he is exceedingly weak. He has had no night-sweats, no abdominal distress of any kind, and even at the present time does not seem to consider himself seriously ill.

Previous History.—He has never had any serious illnesses except the usual diseases of childhood. He denies having had any venereal infections, nor has he ever been subjected to any surgical operations. He is unmarried and his family history is substantially negative, both his father and mother having died in extreme old age. He has several sisters living and well, one brother living and well, and two sisters dead of causes related to childbirth.

General and Negative.—No abnormal pulmonary symptoms can be elicited and he has no palpitation of the heart or cardiac distress. As to gastro-intestinal trouble he denies any kind of indigestion, has never had nausea or vomiting or any acute attacks of pain. His bowels have been regular and he has rarely had occasion to use cathartics. Questioning fails to elicit any abnormalities of the genito-urinary system.

Habits.—He has been a heavy drinker of both whisky and beer for many years, drinking about all that he could get. In addition, he is a heavy smoker. He denies the use of drugs.

Physical Examination.—The patient is fairly well developed, apparently in early middle age, and the most striking thing noted about him on casual examination is the marked difficulty in breathing. He walked into the hospital and into the ward, and since coming in has been able to be up and around, and, as a matter of fact, wanted to get out of bed. While he has not been under observation sufficiently long to enable us to judge his mental condition very accurately, he seems at times a little disoriented.

Head.—Shows no especial abnormalities. Eyes: The pupils are small, but equal, and do not react to light very well. Mouth: Teeth are in fair condition, but the gums show a marked ulceration, with some hemorrhages. The tongue is heavily coated and the breath is foul beyond almost any description. It reminds one of the odor from a pulmonary gangrene. The tonsils are

large and show a number of small areas which do not appear to be enlarged follicles, but have rather the appearance of small, gangrenous areas. On the posterior wall of the pharynx and the palate and on the cheeks are to be seen small whitish superficial ulcers of the same general appearance. The grayish coating of these ulcers cannot be wiped off, and they, too, give one the impression of small gangrenous areas. A considerable quantity of dark bloody mucus is in the mouth.

Neck.—There is a moderate adenopathy involving both cervical chains and the submaxillary glands as well. The glands are not matted together, do not seem to be especially painful on pressure, and the largest of them is hardly as large as the end of one's thumb. There are no abnormal pulsations or tumor masses.

Chest.—The external visual examination of the chest shows nothing special. Over the right lower lobe posteriorly there is diminished tactile fremitus, slight dulness, and the breath sounds seem a little harsher than normal. I shall anticipate a little to say that this is probably due to the pressure of the liver, which is distinctly enlarged. The apex-beat of the heart is not visible, but can be felt indistinctly in the fifth intercostal space inside of the nipple line. The borders are approximately normal. The cardiac tones are not easy to hear because he has numerous loud moist râles in that part of both lungs near the heart. The pulse is regular, of very poor quality, and the blood-pressure 82/46.

Abdomen.—The abdomen seems to be a trifle enlarged. On palpation we find no tender areas except over the left lobe of the liver, which the patient states to be somewhat tender. The lower edge of the liver can be felt about a fingerbreadth below the costal margin in the nipple line and about 3 fingerbreadths below the ensiform. The spleen is just palpable and seems only moderately hard. The kidneys cannot be made out on palpation and no abnormal masses can be made out. The genitalia are negative.

Extremities.—The upper extremities show a little cyanosis and the glands of the axilla are slightly enlarged, but not hard. The lower extremities show nothing abnormal except for a small

subcutaneous tumor on the right thigh—evidently a small lipoma. In particular, there is no edema. The reflexes are all sluggish and neither the abdominal nor the cremasteric or patellar could be obtained.

I have read you the history as it was taken early this morning when the patient entered the hospital, that is, five or six hours ago. Since coming into the hospital two symptoms of importance have developed. The first and most important of these is that he has developed a number of cutaneous hemorrhages. As you see, these hemorrhages are most marked on the abdomen, the insides of the thigh, the insides of the calves, and, to a less extent, on the neck and face. They vary from pin-point size to the size of the palm of one's hand. There can be no question at all about their being straightforward cutaneous hemorrhages, and the interesting feature about them is their very sudden development while the patient was under observation. Indeed, I did not know that they existed until glancing over the patient just before coming into the clinic. The second important symptom is that the patient is bleeding very freely from the mouth. I have tried in vain just a few moments ago to find a bleeding point or points, but it seems to ooze from the mucous membrane all over. It does not seem to come from the gums any more than from the buccal mucous membrane. In addition to these symptoms my intern tells me that the patient is vastly weaker than when he came into the hospital this morning, and he now appears to be a very ill man, although still able to walk around.

I think perhaps the most instructive way to take up this case is to discuss it first as it appeared to the house physicians this morning before the cutaneous hemorrhages developed, because this latter symptom makes the condition somewhat plainer. Going back to the history for a moment we find nothing which gives us any special clue to the patient's condition in his history. He was well until a week or so ago. His only complaints were shortness of breath and cough, coupled with pain in the right side. The examination made on entrance and repeated by me just now fails to show any pathologic condition in the lung capable of accounting for these symptoms. This mode

of onset does not fit in at all with an acute tuberculosis. Pneumonia is hardly to be considered, especially in view of the fact that the patient's temperature is only subfebrile, not having gone above 100° F. For all that, the lung condition was the thing which impressed the physicians in the examining room, since he came to the ward with the diagnosis of a pleural effusion on the right side. Guided by this tentative diagnosis and before examining the patient carefully the junior intern made an exploratory puncture of the right pleural cavity, but had only a dry tap.

Our examination of the heart is not very satisfactory because of the many large râles in the lungs in the neighborhood of the heart, but so far as we can make out there is no abnormality in the heart itself. I may say that my intern has just informed me that a fluoroscopic examination was made, the report of which has not been delivered as yet, but no abnormalities were found in the chest. A blood-culture was taken immediately on entry, but we shall not know the results of this for another twenty-four or forty-eight hours.

The examination thus far leaves us, then, almost without a clue as to what might be the matter with our patient, were it not for the conditions which obtain in the mouth. If we analyze them as I read them to you a few moments ago, you can see that they consist substantially of a severe stomatitis with small areas, not much bigger than large pinheads, of what is apparently gangrene. These are in addition to large inflamed tonsils and some hemorrhage from the mouth. These findings should, I think, direct our attention to a certain group of diseases which are quite prone to begin with mouth lesions. I refer in particular to scurvy, purpura hæmorrhagica, and acute leukemia. Suppose that we consider these possibilities and see if we can come to any conclusion. First, as regards the rapidity of onset, we must say that while scurvy sometimes starts in fairly acutely, I have never known a case of scurvy to develop to any such extent as this in a week. Our questioning of the patient has not been very satisfactory in regard to his previous diet, and especially relative to his getting sufficient quantities of food con-

taining the antiscorbutic vitamin, but we get the impression that he was on a pretty satisfactory diet. One point, however, I should like to emphasize because it seems to me to be the important one here, and that is, that the gums are not characterized by an especial sponginess. Now the gums in scurvy are, in my observation, invariably spongy. Hemorrhages may occur in the gums in purpuric and leukemic conditions and in scurvy too, but the essential point in the latter is the sponginess. I sometimes compare it with the old-fashioned red plush covering one used to see in sleeping cars. I do not know how to account for the small necrotic areas if this were a case of scurvy. As we regarded the case this morning we did not consider seriously the diagnosis of purpura hæmorrhagica since the amount of bleeding from the mouth and gums was too small at that time to suggest this diagnosis. As we see the case now we must, however, take the differential diagnosis of purpura very seriously. Purpura frequently has mouth symptoms, indeed, they are quite common in this disease, although areas of gangrene I do not remember to have ever seen. Laying aside this diagnosis for a moment let us consider the third possibility, namely, acute leukemia. This disease starts in very commonly with mouth symptoms, indeed, in a considerable number of cases which I have seen I should say it was by far the commonest mode of onset, and it is in this disease in particular that we find gangrenous processes about the mouth involving even the tongue. Hemorrhages are exceedingly common, indeed, I should say almost as common as in purpura. In general, acute leukemia is a febrile disease, although in a number of cases the fever is low. The general impression one gets of this disease is rather that of an acute infection of some sort. If we ask ourselves whether the hemorrhages which have developed since morning speak for or against an acute leukemia, I think we must say they are perfectly compatible with this diagnosis, although perhaps somewhat more frequent in purpura.

On the basis of the symptoms alone and the examination as revealed thus far, I think a certain diagnosis is quite impossible. On the other hand, as a result of the laboratory work which we

have done the diagnosis has been cleared up. The laboratory work I refer to is the blood examination which disclosed a rather unusual state of affairs. The red corpuscles are 5,300,000, the hemoglobin 95 per cent., and the white corpuscles 308,000. The differential count of leukocytes which was very elaborately and carefully carried out showed 97 per cent. small mononuclears, 3 per cent. polymorphonuclear neutrophils, and no other cells were found in a count of 300. The oxidase reactions were negative. The reds show a surprisingly normal appearance. The two points which, of course, at once attract our attention are the almost ideally normal red count and hemoglobin and the enormous leukocyte count. I think it probable that the explanation of the high red count is partially to be found in a concentrated condition of the blood, and perhaps also in the fact that the whole condition is so tremendously acute that the toxic factors have not yet had time to affect the erythrocytes to any material degree. However unsatisfactory this explanation may be, I can give you no better one. With the white count as we have found it the diagnosis is cleared up at once. We have a typical case of acute lymphatic leukemia of the small-cell type. High as are the counts in this disease, this count is distinctly high. It is quite generally the case that the count in acute leukemia runs up rapidly with each succeeding day and week of the disease. Indeed, it is not at all infrequent to find that the first blood-count may show an actual leukopenia, and then within two weeks to have counts of 100,000 or 150,000 whites. As a rule, the lymphocytes are of the large variety, so that this case is unusual in this respect.

There is, however, in spite of the apparent simplicity of the diagnosis, one thing to be borne in mind, and that is the possibility of the whole disease being due to an acute septic process starting from the mouth. When I say "septic process" I mean other than that of an acute leukemia which I personally believe to be an infectious process, although it is only fair to say that it is not considered so by many, perhaps even most of those who have written upon the subject. This is a good place to put on record a case which is very much in point.

A child of twelve years was taken sick with some acute infectious process. At the end of a week or two, the child not appearing very ill, a distinguished pediatrician was called to see the case, who had his assistant make a blood examination, finding, I am told, 180,000 whites, with a very great predominance of lymphocytes, mostly large. Doubting the correctness of the examination, he had a second one made, with substantially identical results, and pronounced the case one of acute lymphatic leukemia, and made the corresponding prognosis, stating that nothing further could be done. I happened to see the case at that time, and repeated the blood-count myself, finding 120,000 whites, with about 75 per cent. large mononuclears. The thing about the child was that it did not seem in the least ill and there was no enlargement of the spleen or adenopathy. The diagnosis was very quickly settled, however, not through any skill on my part, but by the fact that the child while I was still at the house had two or three perfectly typical paroxysms of whooping-cough. I then remembered that Cabot had put on record a case of this sort, and told the family that I did not believe that it was a case of leukemia, which diagnosis proved to be correct by the child running a typical case of pertussis, and with perfect recovery. A few days after the time I was called in consultation and while the whooping-cough was at its height the count had dropped to practically normal figures. In addition to this case I have seen a count of over 100,000 with a great increase in the large mononuclears in a straight septic process in which there was no suspicion of leukemia. However, in spite of these facts the case before us is so characteristic when taken in connection with the blood-picture that we have not the least hesitation in stating that it is a perfectly characteristic leukemia of the small-cell type.

Subsequent Note.—The subsequent course of this case was even more rapid than usual. We had not expected the patient to die for perhaps a week or at least some days, but our judgment was distinctly faulty, for death occurred within twenty-four hours. An autopsy was held and the salient features were not different from those which we had found in life. There was an

acute enlargement and hyperplasia of the spleen and liver, of the glands of the neck, and, to a slight extent, of the chest and abdomen. Aside from this there were no features of special interest.

PURPURA HÆMORRHAGICA

THE next case is that of a painter, a Lithuanian by birth, twenty-seven years of age, who came into the hospital yesterday afternoon, complaining of the following symptoms:

Present Complaint.—Bleeding from the nose, great weakness, small discolored areas over the greater part of the body, one arm and one leg, sore throat, and "red urine." These symptoms have been present for about four days.

Onset and Course.—The patient, who speaks fairly good English and is both intelligent and anxious to co-operate, says that he took sick on last Tuesday morning, that is, six days ago, with a sore throat and headache. Up to that time he was in perfectly good health and had felt able to work even the day before. On Tuesday night, toward midnight, his nose began to bleed. He thought he had caught cold in his nose as well as his throat, so he just put some cotton in his nose, which stopped bleeding under this treatment, and went to bed and forgot it. The next morning he got up to go to work and noticed small spots all over the body, ranging from the size of a pinhead to that of a split pea. One or two were somewhat larger. He thinks that they have gotten somewhat fewer, or, at any rate, he says they have become more yellow than they were. Epistaxis has been present every day since it started. On Friday night his nose bled more or less all night. On Saturday afternoon, it having only stopped for short intervals, the patient called a physician, who packed the nose. In spite of this he says it has bled around the packing, and the physician at the examining room states that it was bleeding when he came in. He is not quite sure when he first noticed the "red urine," but is quite confident that it was at least as far back as Thursday, Friday, and Saturday, that is, the three days previous to his admission, which was on Sunday. Since yesterday he has had an intense headache, which was not present before that time.

He has been getting weaker rather rapidly since the onset of the disease.

General and Negative.—On inquiry we elicit a little dizziness yesterday. He states that he had no dyspnea and has not coughed up any blood from the lungs. He has never had any abnormal circulatory or gastro-intestinal symptoms and no abnormal genito-urinary symptoms except the "red urine" already described.

Previous History.—The previous history contains nothing which has any special bearing on the case except that he had rheumatism two years ago.

Family History.—The patient knows very little about his family, and his statements were so unsatisfactory that they were not incorporated in the history.

Physical examination reveals a very anemic looking white male in a good state of nutrition, who appears acutely ill and who is very weak.

Head and *neck* show nothing abnormal except for numerous small hemorrhagic areas on the neck. Pupils are equal, react to light and accommodation, and the left eye shows a fair-sized subconjunctival hemorrhage.

Mouth.—There are no areas of an inflammatory or necrotic nature, but on the inner side of the left cheek is a hemorrhagic area the size of a nickel.

Teeth and Gums.—The teeth were remarkably good for a patient in his station in life, quite different from the usual dirty teeth in our patients here. In particular, there is no pyorrhea and no sponginess of the gums. There are a number of spots from which blood can be seen to be oozing; in other words, the gums are bleeding, but are neither spongy nor ulcerated.

Nose.—We cannot examine this because of its being packed, but, as you see, there is some oozing alongside of the gauze from the right nostril.

Chest is well developed and shows hemorrhagic areas ranging from a dull red to a deep inky blue all over. These, as you see, do not change on pressure, and they vary in size from that of a pin-point up to 3 or 4 inches in diameter.

Lungs show no abnormalities.

Heart.—The apex-beat is in the fifth intercostal space, inside of the midclavicular line, of normal width. The heart boundaries are substantially normal and on auscultation one hears a well-defined systolic murmur at the apex, not transmitted to the axilla. The second pulmonary is not accentuated.

Abdomen.—The skin is covered with hemorrhagic areas. The spleen is not palpable. The liver dulness is normal, and no abnormalities can be detected anywhere in the abdomen.

Genitalia are negative.

Extremities are negative except for numerous hemorrhages.

The present condition of the patient is substantially that in which he entered the hospital, except that he is distinctly weaker. The senior intern made the following note this morning:

"Heart: Rate is rapid; systolic murmur at the apex seems very loud.

"The skin shows a considerable increase in the number of hemorrhagic spots which were there last night. The patient's temperature on entering the hospital was 105° F. by rectum, with a pulse of 100 and respirations of 30, and is now over 104° F." He further noted that neither liver nor spleen was palpable and because of the old history of rheumatism two years ago and the rather loud systolic murmur at the apex, he wrote as his tentative diagnosis, "Acute exacerbation of an old endocarditis."

Now let us review this case a little and see if we can pick out the salient points which have a diagnostic bearing. To begin with, we have a young, strong, healthy individual who declares positively that he was perfectly well and able to do laborious work until a few days before his entrance into the hospital. Although he states he had rheumatism a couple of years ago, further questioning elicits the fact that the rheumatism consisted of some rather vague pains in the joint, and it only incapacitated him for work for one day. He does not think he had any fever and, on the whole, his statements do not carry the least conviction that he had a general attack of polyarthritis rheumatica. Furthermore, he gives no history of

having had any symptoms referable to the heart. No shortness of breath, no edema of the ankles, no palpitation, etc. This makes it very improbable, to say the least, that he actually has an organic heart lesion, and our physical examination of the heart does not disclose any especial hypertrophy. The murmur at the apex is common enough after hemorrhages and does not, in my judgment, demand that we make a diagnosis of endocarditis. Furthermore, the very sudden onset and the very pronounced hemorrhagic tendency is, in my opinion, out of all proportion to anything which we might expect to find even in a very severe septic endocarditis. It is true that we find small embolic hemorrhages in the skin and hematuria in this latter condition, but the other symptoms are, in my estimation, not to be satisfactorily accounted for by the assumption of an endocarditis.

The question which we must put to ourselves is this: What conditions give rise, in a previously healthy, strong individual, to hemorrhages all over the body, to hematuria, to severe epistaxis, with high fever, and at the same time give a practically negative physical examination of the internal organs? We think at once of the so-called hemorrhagic diseases and of the acute infections in which there is a strong hemorrhagic tendency. We may dispose of these latter, I think, very quickly, for there is no evidence of anything like variola or scarlatina or similar infection in which we sometimes get extensive hemorrhages. We must think, then, first of purpura hæmorrhagica and of another disease which quite frequently starts in exactly this way, namely, acute leukemia. Without further investigation we cannot make the diagnosis. Our laboratory reports show that the Wassermann is negative, the urine, as would be expected, contains albumin in large amounts, due to the large quantity of blood present, and now we come to the most important of all—the blood. This shows about 2,700,000 red corpuscles, 37 per cent. hemoglobin, and the most remarkable white count which I personally have ever seen, namely, 250 per cubic millimeters. This extraordinarily low white count struck us as so very unusual that it was repeated after a few

hours, and found to be absolutely correct. I have myself examined the stained specimen of blood, which is equally remarkable. To begin with, the reds look to be in fairly good condition. There is no special variation in size and shape, and they do not suggest the severe anemia indicated by the red count and by the 37 per cent. hemoglobin. A half-hour's examination of the smear failed to show more than one or two white cells, which were small lymphocytes. Now one must not forget that this can occasionally happen when the cells congregate around the edges of the specimen, and to assure myself that this is not the case I have gone over the edges of the preparation systematically, but with no better luck. Leukocytes are as scarce as the proverbial hen's teeth.

A blood-culture was taken immediately on entrance, which has remained sterile up to the present time. On the basis of this blood examination are we able to draw any further conclusions? I think so. It at least enables us to rule out with a fair degree of certainty an acute leukemia. This disease, as you all know, starts in sometimes with symptoms almost exactly similar to those which this patient presented. However, certain features are lacking. We found no severe ulcerative processes about the mouth, no enlarged tonsils or gingivitis, and, in particular, we found no enlargement of any glands or of the spleen. You might suppose that the leukopenia alone would suffice to rule out an acute leukemia, but this is, unfortunately, not the case, for we quite frequently have a distinct leukopenia at the beginning of an acute leukemia, although I have never known of a count similar to the one before us. The next thing which I would think of would be that this is possibly a case of very acute scurvy. For this, however, we have no favoring data. The gums are not spongy and not ulcerated and the man was earning a good living, eating, according to his statement, good food. In particular, we do not find any evidence of the scurvy sclerosis, a symptom which almost certainly would be present in the presence of so many hemorrhages were this a true scurvy. So we drop this from the list of diseases to be considered. This leaves us with the diagnosis of purpura hæmor-

rhagica uppermost in our mind. The symptoms and general course of the disease are perfectly compatible with this diagnosis, but, on the other hand, we need confirmatory data. Now in order to make a diagnosis of purpura hæmorrhagica one should have at least the following: Outspoken hemorrhages, a low red count, an approximately normal coagulation time, with a distinctly non-retractile clot, and, above all, a distinct diminution in the platelet count. We will check these up seriatim. Our coagulation time was a trifle longer than normal, but not materially so. In my judgment it is of little value to give the actual number of minutes, since this varies so greatly, depending upon the method used. My own preference is to always compare it with a known normal, and then it is of little importance which method is used. The platelet count showed an enormous diminution, there being but 25,000 per cubic millimeter. I may say that this count necessitates counting everything that resembled a platelet, and it is more likely to be too high than too low. In our smears stained by the Wright method, which ordinarily shows the platelets very well, we found them extraordinarily scarce, thus confirming our very low actual count. The bleeding time, I regret to say, was not accurately noted, but it was noted that the cut bled much longer than commonly, so I think we are safe in saying that there is a greatly prolonged bleeding time.

Now just a few words on the subject of purpura hæmorrhagica and the allied conditions. The diagnosis of these is still far from satisfactory. Our case presents every feature of an extremely severe case of this malady and only one point strikes our attention. We would rather expect a slight leukocyte increase as a result of the hemorrhages and, instead, we find this extraordinary diminution. However, I think the septic element in the case may explain this without doing violence to our diagnostic considerations. We are still ignorant of the real nature of purpura hæmorrhagica. A case such as ours impresses us very strongly with the idea that it is an acute septic process and, indeed, many cases are on record, generally under the name of acute septic or hemolytic anemia, in which the case was

regarded as one of sepsis and the hemorrhages as being of a secondary nature. I know of absolutely no criterion which will enable us to separate these two types of cases, and it may be, after all, that they are really identical and that purpura is itself a septic process in some cases.

You may ask, Is not this an extraordinarily rapid course for a case of acute purpura? and I can only say that while it is a rapid course it is by no means unusually so. There is another type of the disease, the so-called purpura fulminans, which is much more rapid. I have seen only one case of this latter, and in this patient the family doctor saw him in the morning, with a slight sore throat and a moderate fever, but by noon his temperature was around 105° F. and he showed a few small hemorrhages in the skin. By suppertime the hemorrhages were quite extensive, and when I was asked to see him in consultation about 11 P. M. that same night the hemorrhages were so extensive that he resembled nothing so much as a blue plum, and he died an hour later. I cannot help feeling that all such cases represent cases of foudroyant sepsis of some very special nature which has a particular tendency to act upon the capillaries and render them permeable, thus producing hemorrhages.

It may not be amiss to call attention to the possibility of mistaking the diagnosis in cases such as these and confusing them with a purpura variolosa. I myself have never seen a case of the latter, and yet I know of an instance a number of years ago in which this mistake was made, and as a result a number of cases of smallpox developed, so it is worth while bearing this in mind in communities where smallpox is frequently found.

Subsequent Course.—The further course of this case was extremely rapid. He died on the evening of the day on which he was shown at clinic, and the autopsy disclosed the following:

Anatomic Diagnosis: Extensive petechial and ecchymotic subcutaneous, submucous, and subserous hemorrhages; generalized anemia; melena; hemorrhage into the left kidney pelvis; hemorrhage in the bladder; slight hypoplasia of the bone-marrow.

In the light of the autopsy have we any reason to revise

our diagnosis? I think not. We had considered the possibility of an extremely rapid case of aplastic anemia, although I had not discussed it with you in clinic. This hypoplasia of the bone-marrow might possibly be construed in that sense, but the course of this case is so unlike the usual one of aplastic anemia that I do not think it should be considered among the diagnostic probabilities.

HODGKIN'S DISEASE (ABDOMINAL TYPE)

THE next patient I wish to present to you is a Pole, fifty years of age, who came into the hospital about two weeks ago. I may note that the diagnosis which was made in the examining room was that of pernicious anemia. My point in making this statement is that while it is, of course, impossible to make more than a cursory examination in the examining room, yet this was the way in which the case impressed the examining physician. He complained of five very definite symptoms: *first*, pallor; *second*, shortness of breath; *third*, cough; *fourth*, swelling of the ankles; *fifth*, marked loss of appetite. The duration of these symptoms he dated very definitely at seven weeks before his admission.

Onset and Course.—The patient, who is unusually intelligent and anxious to co-operate, states that he was a perfectly well man with ruddy cheeks and strong muscles up to about seven weeks ago. He thinks he may have had a little swelling of the ankles, in the evening particularly, a week or two sooner. Then he commenced to notice a slight, sticking pain which traveled from the left hip to the left inguinal region, and then into the left testicle, where the pain assumed a clamping nature. He paid but little attention to this pain, thinking it was some minor bowel trouble, and went on about his work as usual. The pain recurred, and it was after two or three weeks of this pain that he suddenly realized that the symptoms complained of by him above had already developed to quite a degree. He was quite struck with his pallor, which was the more noticeable to him because of his previous ruddy complexion. The shortness of breath he had not noticed except when he exerted himself, but by the time he was ready to enter the hospital he was unable to walk upstairs except with great difficulty. The cough came on rather insidiously and now exists with very little expectoration. There has never been any blood in the sputum

or any special pain in the chest. The swelling of the ankles has been growing steadily worse and he notices it only in the evening. It is worthy of note that it disappears if he lies down even for an hour. His loss of appetite has been so severe in the last three or four weeks that he has eaten but very little, and he says that everything he eats tastes like a rag. He complains of abdominal distention after eating or drinking, even a small quantity. He has had no pain in the abdomen, no nausea or vomiting, and has never noticed anything abnormal in the stools. In particular, there has been no evidence of hemorrhage. He has complained of numbness and tingling of the hands and feet, but this has not especially disturbed him.

Past History.—Aside from the acute articular rheumatism fourteen years ago, he has had no medical or surgical conditions worthy of note. He drinks alcohol to a moderate extent, but has not done so for a number of years. A doubtful history is had in reference to lues, but I may say that his blood Wassermann is absolutely negative.

The family history is quite extensive, but without any especial bearing upon his case.

General and Negative.—We find no nervous symptoms complained of except that he has lately been very dizzy when sitting up suddenly. He does not think he ever had any fever, but has noticed rather severe night-sweats in the last few weeks. There are no genito-urinary symptoms of importance, and he has no especial complaints on the part of the special senses.

Physical Examination.—The patient looks to be about fifty, is strikingly pale, and yet fairly well nourished. He is perfectly oriented and does not give the impression of a patient very acutely ill.

Head and Neck.—The left pupil is a trifle larger than the right, but both of them react well to light and accommodation; no evidence of jaundice in the conjunctiva. The ears, nose, and throat are negative, except that he has poor teeth so characteristic of most of our County Hospital patients. The tongue is moderately coated, the pharynx pale, the tonsils small, and showing no evidence of disease. The neck shows no abnor-

malities, and in particular there are no glands palpable in the cervical or submaxillary chains.

Chest.—The lungs show no abnormal findings except an occasional râle at the apices.

Heart.—Apex-beat is in the fifth intercostal space well inside the nipple. The boundaries are those appropriate for a man of his general build. Following the first sound at the apex there was a soft, short systolic murmur which remains quite localized to the apex. The second pulmonary sound is not accentuated. The pulse is of good volume, regular, with a systolic pressure of 100 and a diastolic of 55.

Abdomen.—As you will see on looking carefully there is a fulness in the epigastrium to the right of the midline, but no definite mass is palpable. The border of the liver can be plainly felt, sharp and somewhat firm, about 2 or $2\frac{1}{2}$ fingerbreadths below the costal arch in the right nipple line. On turning the patient over in a semilateral position one can feel the spleen, indeed, almost see it, about 3 fingerbreadths below the costal border. It is quite hard.

Genitalia.—These disclose no abnormalities.

The upper extremities show a rather interesting clubbing of the fingers which the patient insists is hereditary in his family. We are inclined to accept this statement at its face value, since we have not been able to find any adequate cause for this clubbing. The lower extremities show a moderate edema of the ankles, which is soft and has all the properties of a tolerably fresh edema.

The reflexes are all normal and there are no areas of anesthesia or paresthesia noted. The patient shows no evidence of any atactic phenomena.

With reference to the blood we may note that in view of the fact that the patient was sent into the ward from the examining room with a diagnosis of pernicious anemia, and in further view of the fact that he is manifestly severely anemic, with a spleen considerably enlarged, a blood examination was made at once, with the following result: Erythrocytes, 2,600,000; leukocytes 7600; hemoglobin 40; color index 0.81. A differ-

ential count of leukocytes showed polymorphonuclear neutrophils 74 per cent., small mononuclears 19 per cent., large mononuclears 6 per cent., eosinophils 1 per cent. Microcytes, macrocytes, and poikilocytes were fairly numerous and considerable polychromatophilia was noted. No nucleated forms, either large or small, were encountered in a fairly careful search. A blood-culture was made, but remained sterile. Urinalysis showed nothing of consequence. An Ewald test-meal was given, and only 10 c.c. was recovered at the end of an hour, which showed no macroscopic abnormality and no evidence of blood, and with a free acidity of 10 and a total of 22. The stools were normal in every respect, both microscopically and clinically.

To begin with, I should like to discuss the diagnosis with you as it appeared at that time. Summing up the symptoms they were manifestly those of some disease starting acutely, producing a severe anemia within a couple of months, with cough, shortness of breath, and edema of the ankles. There is nothing in the cardiovascular or renal systems to account for any of these things. The heart borders are normal and the little systolic murmur at the apex is to be interpreted, in my judgment, as an accidental murmur. I think you will all agree with me that the first thing to be thought of was pernicious anemia, and in favor of this diagnosis one could adduce the large, hard spleen, which, while not always present in pernicious anemia, is a valuable confirmatory sign if found, the severe anemia, the absence of leukocytosis, and the relatively slight loss of weight. On the other hand, we notice that the differential count showed 74 per cent. of polymorphonuclear neutrophils with a color index of 0.81, and these two findings hardly fit in with the assumption of a pernicious anemia. The thing which impressed me most at this time on going over the case was the unquestioned enlargement of the liver. I was unable to make this fact fit into the clinical picture of a pernicious anemia. My first thought would have been that of a malignant tumor somewhere in the abdominal cavity, with metastases in the liver. Of course, the liver seems to be uniformly enlarged, but when one remembers how small a portion of the surface of the liver is accessible to

palpation it is readily comprehensible that there may be large numbers of metastases in the liver and yet not a single one of them be accessible to the palpating fingers. To this assumption, however, could be opposed another objection, namely, that it was extraordinarily difficult to account for so much of an enlargement of the spleen unless we consider that the postulated tumor was primary in the spleen. It had been my fortune to see one or two primary splenic tumors, and I revolved this possibility very carefully in my mind, without being able to arrive at any definite decision. After having the patient very thoroughly purged, a second physical examination of the abdomen revealed a small mass low down in the left lower abdominal quadrant and situated very deeply. While of small size, apparently not much bigger than a duck egg, it moved a little bit, but not nearly enough to suggest its being situated in the bowel. It felt like a conglomerate mass of several smaller tumors. About this time a second blood examination was made, showing this time nearly 3,500,000 red blood-corpuscles, 45 per cent. hemoglobin, with a color index of 0.70, with a white count of 4500, and almost exactly the same differential count as was observed on the first examination. Inasmuch as the patient was growing definitely worse each day, we attributed the apparent increase in the reds and hemoglobin to a concentration of the blood depending upon insufficient fluid intake and free purgation. A careful rectal examination was made, which elicited nothing abnormal.

Roentgenologic Examination.—The report from the barium clysma was substantially negative. Filling occurred without any delay, with good tone of the bowel and no filling defects. The examination of the stomach and small bowel with the barium meal was negative so far as any evidences of pathologic changes in the gastro-intestinal tract were concerned.

It had been noted on the first examination that the inguinal glands were a little enlarged, and in the week after his admission to the hospital they enlarged quite definitely. I call your very special attention to this fact, since in my judgment it is practically the most significant confirmatory fact we have elicited. Inas-

much as the blood examination made leukemia out of the question and made pernicious anemia more than doubtful, we were now faced with this question: What could there be in the abdominal cavity producing a very decided enlargement of the spleen, a moderate though very evident enlargement of the liver, and at the same time produce a definite and increasing enlargement of the inguinal glands, more on the right side than on the left. There seemed to be two possibilities only, or at the very least two possibilities presented themselves which seemed far and away the most likely. They were, a malignant tumor of the abdomen or a very unusual case of Hodgkin's disease. It then occurred to us that there might be an enlargement of the bronchial and mediastinal glands and that the entire abdominal picture might be secondary to these. To determine this we ordered a roentgenologic examination of the chest, which was made, with the following result: The lungs showed a little increased density in the upper right lobe, suggesting an old tuberculous focus. The markings at the hilum were slightly increased, but hardly sufficient to warrant a statement that there was definite enlargement of the glands. We could, therefore, derive no assistance from this source.

In the meantime the patient's condition was slowly but steadily growing worse. He showed a little fever in the afternoons, not often higher than 100° F., but nearly always present.

Weighing the two diagnoses above suggested, the one against the other, we were confronted with the fact that it was difficult to explain the enlargement of the spleen if we assumed that there was a malignant tumor concealed somewhere in the abdomen. On the other hand, if we assumed that the enlarged spleen and the few inguinal glands were a Hodgkin's disease, it was difficult to explain the enlargement of the liver, since this seemed out of all proportion to the acuteness of the process and to the size of the spleen. We considered all of the probable sites for primary malignant tumor, such as the pancreas, prostate, colon, vertebral column, small bowel, and we could find no symptoms warranting such a diagnosis. Under the circumstances the obvious thing to do was to remove one of the

rapidly enlarging inguinal glands and subject it to microscopic diagnosis. This we were more inclined to do since we had come to the conclusion that the small mass we felt low down in the left iliac quadrant was made up of a number of small glands.

As I recite the details of this case to you it may seem to you the very simplest thing in the world to make a pathologic examination from the section of a gland. Sometimes it is a fairly simple matter, but I have seen not once but many times a gland subjected to microscopic examination which elicited two or three different diagnoses from pathologists of great experience and reputation. In this case after a careful study of the gland section the three pathologists to whom the sections were submitted were unanimous in the diagnosis of Hodgkin's disease. I have myself no opinion to offer on the subject in view of the fact that several infinitely more expert opinions are available. Shorn of all the technical details the report on the gland examination states that there was no evidence of fibrosis, caseation, or necrosis, and that it was a typical Hodgkin's granuloma extremely active.

With this examination the case seemed to be definitely disposed of and there are now but few additional facts of moment to report. While the patient is not losing weight rapidly, he is growing paler day by day, his weakness is increasing very markedly, and now and then he is a trifle irrational. Despite repeated careful examinations of the abdomen we are unable to find any additional tumor masses, and the liver and spleen stay about the same size as when he entered the hospital.

It may not be amiss in view of the very distinct rarity of a case of this kind and in view of the fact that the diagnosis can be now made with certainty, to discuss this type of Hodgkin's disease a little. As you all know, the cervical glands are almost always enlarged very early in the disease, and one seeing many cases of this malady gets accustomed to looking for them here first of all. I am not prepared to say off-hand just what proportion of cases show as the initial symptom and enlargement of the cervical glands, but it is certainly quite a large proportion

of them. Next in number, if studied carefully from the beginning, are the patients showing an involvement of the mediastinal glands as the initial symptom. These are the patients who come under observation with coughs, sometimes aphonia, distended veins on the chest wall, shortness of breath, and other signs of increased intrathoracic pressures such as one commonly finds with good-sized aneurysms. In the absence of enlargement of the peripheral glands the diagnosis is scarcely possible in any other way than as the result of the *x*-ray examination of the chest. However, a little time generally brings with it the enlargement of the axillary or cervical glands in addition to those of the chest, and then the diagnosis is fairly clear.

There is a still further group of cases in which the disease seems to originate in the spleen, the so-called anemia splenica of the older writers. There is very little to be said about this group of cases except that a diagnosis in the absence of enlargement of numerous gland groups would rest on rather slim evidence. There is still another group of cases, known as the Pel-Ebstein group, which is characterized by remarkable intermittent pyorexia. In one patient whom I saw some years ago, himself a physician and who had observed himself most accurately, the periods of fever lasted exactly fourteen days, with exactly fourteen-day intervals between the attacks. Of these attacks he had had six or seven before I saw him and he had six or seven more before his death. Each attack was synchronous with a simultaneous enlargement of the liver and spleen. The mediastinal glands became greatly enlarged and ultimately the peripheral glands as well.

There would seem to be no inherent reason why the enlargement of the glands in the majority of cases of Hodgkin's disease should begin with the cervical chains. It is, however, a well-known fact that the onset with enlargement of other groups is quite rare, and in particular the enlargement of the retroperitoneal glands as an initial symptom is exceedingly rare. This case must, I think, be placed in this latter category, since the most painstaking examination fails to reveal any glands outside the abdominal cavity with the exception of those in

the groin, which are scarcely larger than the end of one's finger, to be enlarged. In the case before us there is no difficulty at all in ruling out a tubercular adenitis beginning in the retroperitoneal glands, since this would not produce the enlargement of the liver and spleen which are the striking features to be found in the patient before us. There are, however, cases in which this differential diagnosis is one of extreme difficulty. In a previous number of these Clinics I reported a case of tuberculosis of the abdominal glands in a nurse which for a long time we regarded as being a case of the abdominal type of Hodgkin's. After many months of illness the peripheral glands became somewhat enlarged, and on removal of one of these it was found to be tuberculous in nature. The patient made a slow but good recovery, and in the several years which have elapsed since then has been in excellent health.

If our diagnosis is correct it carries with it a fatal prognosis, since Hodgkin's disease is fatal. If the patient is still alive at our next clinic I shall show him to you again.

One week later: I wish to report to you the outcome of the patient with Hodgkin's disease whom I presented at the last clinic. In spite of the fact that he seemed to be in fairly good condition, he died very suddenly and some time before we expected. The postmortem was made by Dr. Singer this morning and these are the organs from this patient. The essential things are, as you see, this greatly enlarged spleen which shows a typical granulomatous infiltration on the cut section. The liver weighs about half again as much as the normal, is smooth and firm, and on its cut section shows the typical granulomatous infiltration. This mass which I show you, and which is made up of a group of iliac glands the size of my fist, was the tumor which we felt low down in the left iliac quadrant. The glands on the right side in the same position are also enlarged, as are also the peri-aortic glands. The mediastinal glands were a little enlarged, but not to an extent to become objects of roentgenologic diagnosis. The lungs, as you see, show a slight tuberculosis apparently healed out at both apices. The heart

valves are normal, as we have postulated, and there are no other significant pathologic findings in the body.

There is little more to be said about the case since the examination of the gland put the diagnosis of Hodgkin's disease beyond doubt. I call your attention to the very decided rarity of cases of this kind. It is, of course, impossible to state whether the enlargement of the spleen, liver, or iliac glands was actually primary, since all three were definitely enlarged, and it is impossible to even make a guess as to which came first. In view of the very rapid course of the disease, an assumption which is borne out by the histologic character of the glands, it is not improbable that the enlargement took place in all of them simultaneously. Indeed, if we think of Hodgkin's as a systemic disease affecting in greater or less degree the lymphatic tissue everywhere, this conception seems most reasonable.

CLINIC OF DR. J. C. FRIEDMAN

MICHAEL REESE HOSPITAL

LATENT JAUNDICE

THIS phrase may be defined as that pathologic condition in which there is an increase of bile-pigment in the blood-serum without a visible deposit in the skin and mucous membrane, a sort of "forme fruste" of icterus. It may or may not be combined with excretion of bile-pigment in the urine. Of course, every case of clinical icterus is preceded by a stage of latent icterus, as the *hyper*bilirubinemia precedes the deposit of pigment in the skin. This is not the group which we shall speak about at this time, but limit ourselves to some of the cases in which, throughout the course of the disease, no visible icteric tint of the skin is present.

It should be remembered that physiologically bile is present in blood-serum in minute amounts (0.3 to 0.5 in 200,000 parts of blood-serum), and that this must be increased eight to nine times in amount and continue at this height for some time before the tissues become visibly impregnated and the kidneys excrete it. When the bile in the blood-serum lies between these two extremes we have the condition defined as latent jaundice.

It is well to recall that Whipple and Hooper have shown that the liver and spleen are not the only sites of bile-pigment formation and, as suggested by them, the endothelium of the blood-vessels may play a part in its production from hemoglobin, and by others that lymph-glands and bone-marrow also are concerned. And, furthermore, that according to Van den Bergh and others, all cases of jaundice may be divided into two main groups: (1) The obstructive or mechanical, and (2) the dynamic group (which corresponds roughly with those cases formerly called

hematogenous). Van den Bergh has proposed a modification of the Ehrlich diazo test for bile-pigment in the blood-serum by which these two groups may be distinguished from each other. Not a great deal has been added to our knowledge of obstructive jaundice in recent years. Eppinger has shown that when obstruction takes place in the finer bile capillaries, bile-stained thrombi are present which aid the obstruction to the outflow of bile, and that in many cases of jaundice due to necrosis of the liver cells the finest bile-passages are torn open, allowing the bile access to the capillary system of the liver. It is in the dynamic type of jaundice that the greatest advance has been made. In these cases it is not a question of obstruction to the outflow of normally formed bile, but apparently first an increased destruction of red blood-cells with a consequent increased formation of bile-pigment (often without increase of other bile constituents, such as bile salts). Furthermore, the pigment is formed by the endothelial structures of spleen, lymph-glands, liver, and other structures, and only because the liver is unable to excrete it all (perhaps because the liver cells are also damaged) does the pigment remain in the blood in increased amounts, giving us a latent or visible jaundice, as the case may be. Examples of this group are the jaundice of pernicious anemia, hemolytic jaundice, the hyperbilirubinemia which occurs during the absorption of hematomas and in like conditions. To sum it up, then, in dynamic jaundice we have increased blood destruction, increased formation of extrahepatic bile-pigment, with resulting increase in bile-pigment in the blood-serum, while in obstructive jaundice we have an escape of normally excreted bile into the blood-stream. These two types are distinguishable by chemical tests. Both of these groups may, however, give rise to latent jaundice, and it is this clinical aspect which we wish to bring out in the following discussion.

In order to detect these cases clinically we must have some simple test for bile in the blood-serum which, while specific for bilirubin, will yet not react with the minute amounts present in the normal serum. Such a one is that of Fouchet, an oxidizing agent consisting of 5 grams of trichloracetic acid, 2 c.c. of 10

per cent. ferric chlorid, and 20 c.c. of distilled water. This will change color with bilirubin solutions between dilutions of 1 : 20,000 and 1 : 60,000.

The method is as follows: Five c.c. of blood are withdrawn from the patient, allowed to clot, and 3 to 5 drops of serum taken up in a capillary tube and mixed on a porcelain dish with an equal amount of Fouchet's reagent. In the presence of a hyperbilirubinemia, a green color appears, reaching its maximum within five minutes, the intensity of the green being, of course, proportional to the amount of bilirubin present. However, to be of clinical value we must assure ourselves first that it is specific for bilirubin. That this is so we determined by using the reagent with solutions of a number of the bile constituents, as lecithin and cholesterol. Neither of these gave a positive reaction. Another important point is the practical determination of the fact that a positive reaction does not occur with normal sera. A series of 89 sera were employed for this purpose. Five only were positive. The diagnosis of 4 of these cases was determined and found to be conditions such as catarrhal jaundice, splenomegaly, pernicious anemia, and acute endocarditis, in all of which cases increased bile-pigment is present in the blood. Three doubtful reactions were excluded. It is safe to assume, then, that the Fouchet reagent will not react with normal sera. The other question is whether or not it is sufficiently sensitive for clinical purposes. In order to determine this comparisons were made with what is probably the most frequently used method for the detection of bile-pigment in serum, viz., the Ehrlich diazo test. Of 92 cases in which the Fouchet was positive, the diazo method was positive in only 71. It is reasonably certain then that the Fouchet test is specific, is sufficiently sensitive, and will not react with normal sera.

As an example of the clinical value of this test we may take the following case[1]: A patient, male, forty-two years of age entered the hospital complaining of pain on the right side of the abdomen. It was continuous and sharp, radiated to the epigastrium and to the right side of the back. It began suddenly

[1] From the service of Dr. A. R. Edwards, Michael Reese Hospital.

ten hours before entrance. Vomiting occurred only once, when he took Epsom salts. Has had previous attacks at intervals for the past five years, lasting two to three days each, and in the intervals has had some gastric distress consisting of fulness and pressure in the epigastrium immediately after eating. There has never been any jaundice. There is nothing of importance in the personal or family history.

On examination the face was flushed, skin and scleræ not icteric. Lungs and heart normal. Right half of abdomen quite rigid and tender, the most marked point of tenderness being about at the level of the umbilicus. No tumor was palpable. Temperature 100.2° to 100.4° F. per rectum. Leukocyte count 14,000, with 77 per cent. polynuclears. Urine showed a trace of albumin and no bile. It was then a question of an infection of the gall-bladder or appendix. No absolute decision could be arrived at. With Fouchet's reagent the blood-serum showed a definite trace of bile. At operation there was found a greatly distended, thickened, and gangrenous gall-bladder, with numerous stones and a slight increase of peritoneal fluid definitely bile stained.

A case, then, of latent jaundice in which the clinical findings were confusing, owing to the low position of the gall-bladder, and in which the diagnosis was made much more probable by the presence of increased bile in the serum.

In order to determine how frequently latent jaundice might be present in pathologic conditions of the gall-bladder, a number of cases (of course without visible jaundice) were examined during and between attacks.[1] There were 7 such cases in which the blood was examined before the colic had entirely ceased, and in whom there was no visible icterus, the urine containing no trace of bile-pigment. The diagnosis of cholecystitis in all but one was confirmed by operation, and in that one the typical history of the palpable gall-bladder left no doubt as to the correctness of the diagnosis. In all the cases stones were present in the gall-bladder or cystic duct, or both, and in 3 there was empyema of the gall-bladder besides. In the other 4 normal appearing bile was present. In every one of these cases there

[1] Report on larger series of cases of cholecystitis at present in preparation.

was a positive reaction in the blood-serum with Fouchet's reagent, the intensity of the green coloring varying from a faint trace to double plus. Apparently there was no fixed relation between the severity of the clinical picture and the intensity of the reaction. In the history quoted above there was a gangrenous gall-bladder with peritoneal exudate and only faint reaction, while other cases not nearly so acute give much more marked reactions. As to the method by which the bile enters the blood-serum in these cases in which only the cystic duct and the gall-bladder are involved, we can only theorize. In the case of which the history is given the bile-stained peritoneal fluid must have come directly from the gall-bladder, as the blood-serum did not contain enough bile-pigment to color the body secretions. On the contrary, the slight increase of pigment in blood-serum may have been due to absorption from the peritoneal cavity. This, however, is an exceptional case. In the others we must assume either a mild cholangitis which has caused a slight obstruction in the finer bile passages, *i. e.*, an obstructive type of jaundice or a hepatitis with changes in the liver tissue giving us the dynamic type of jaundice. Both of these conditions have been demonstrated histologically in sections of livers of such cases. The fact that the empyema cases often gave marked reactions leads one to believe that inflammation of the bile passages is of importance in this condition. As to the earliest period in which the reaction appears I cannot say. The blood of one patient was examined within forty-eight hours and was positive. Whether or not it occurs earlier I do not know. Another question of interest is whether or not such patients would go on to a definite visible icterus. Of the 6 cases operated on, no evidence as to the future course could be given except the negative point that the common duct was not involved. The one case not operated recovered completely without visible jaundice. A second blood-serum taken ten days afterward, when the acute symptoms had almost completely ceased, gave a negative Fouchet test. It is certain, then, that a patient showing a positive bile reaction in the serum will not necessarily go on to a visible jaundice.

If the increase in bile in the blood-serum in cases of cholelithiasis and cholecystitis is the result of an ascending inflammation, one would expect fewer positive reactions in patients examined between attacks, when the inflammation is probably again limited to the gall-bladder, the symptoms being indefinite abdominal pain or gastric distress, and so forth. Nine such cases were examined, of which 4 were positive. These, of course, were not strictly interval cases, as some symptoms were present. In general, one may say that during an attack even without visible jaundice a very large percentage of cholecystitis cases will give a positive bile reaction in the serum, and that when the attack has materially subsided the reaction remains positive for a considerable period of time in a certain number of cases (about 40 per cent.). It is unnecessary to state that in all cases of cholecystitis with visible jaundice with or without pain (of which there were 11) the Fouchet test was always strongly positive.

Another type of case in which latent jaundice may occur is that group of intestinal infections commonly called catarrhal jaundice. The jaundice may be latent from one of two reasons—either it may be an extremely early stage of a more intense visible jaundice, or it may be a very mild infection never intense enough to reach the latter stage. An example is the following: The patient, twenty-nine years of age, complained that for the past ten days he felt drowsy, tired easily, had almost complete anorexia, and slight epigastric distress after eating. He had had no diarrhea, or any recent upper respiratory infection (there was at that time an epidemic of such infections). He had always been in good health except for an attack of catarrhal jaundice five years before, and volunteered the information that the present symptoms were so like those that he had experienced at that time that he had been watching his skin for jaundice. The examination showed normally colored skin and mucous membranes. Chest normal. No tenderness in abdomen. Liver and spleen not palpable. Temperature 98.2° F. Leukocyte count 7600. Urine normal except for slight increase in urobiblin, but no bile-pigment. The blood, however, gave a very definite

reaction with the Fouchet reagent. No jaundice developed, and in the course of the following week all symptoms disappeared. A "forme fruste" type of catarrhal jaundice then. In this connection it is of interest to consider if mild cases of catarrhal jaundice are not of more frequent occurrence than is ordinarily believed, and particularly if the old conception of "biliousness" may not have a firmer foundation in fact than we at present believe, a certain percentage at any rate being slight infections of the upper intestinal tract with latent jaundice.

There are a number of other conditions in which the presence of latent jaundice is of not infrequent occurrence. Severe cases of jaundice following the administration of salvarsan and its derivatives is not uncommon. The blood of various patients to whom arsphenamin had been given twenty-four hours earlier and who had not developed jaundice or, indeed, any untoward symptoms was examined. In 17 such cases[1] a positive reaction was obtained in 6, or 35 per cent. In a study of this question Chargin and Orgol (Archives of Dermatology and Syphilology, vol. 7, No. 4), using a different test, found an increase in bilirubin in the serum of such persons in about 16 per cent. Whether their reaction is less sensitive (as I believe) or whether my series is too small from which to draw these conclusions, I do not know. Certain it is that a positive reaction in 35 per cent. of cases showing no jaundice was unexpected, and demonstrates that liver damage is much more frequent than is ordinarily believed to be the case.

Another condition in which visible jaundice appears with varying frequency is lobar pneumonia. It was thought that here, too, latent jaundice might be rather frequent. In 5 cases examined an increased bilirubin was found in 2. This is a lower percentage than is ordinarily given. These patients were examined after the disease had been in progress several days, so no statement can be made as to the earliest time at which the increase in bile appears. The determination of this point would be of importance, as the early cases of right lower lobe pneumonia are the most difficult to distinguish from acute chole-

[1] These cases were from the clinic of Dr. Erwin P. Zeisler.

cystitis, as in the latter condition we know that the hyperbilirubinemia may be of early occurrence.

In chronic decompensated hearts increase of bile was found in 3 out of 10 cases. In one, carcinoma of the descending colon, the test was positive before operation and negative four weeks later.

Pernicious anemia is a representative of the dynamic group of jaundice cases in which apparently the increase of bile-pigment in the serum, and of its reduction products, urobilin and urobilinogen, in stool and urine are evidences of increased blood destruction and extrahepatic bilirubin production. In 10 such cases examined twenty times the reaction was positive fifteen times. The blood from the same patient varies from strongly positive to negative according to his general condition, as evidenced by the following examples:

Mrs. M.:

12/14/22: Hbg., 60 per cent.; red blood-cells, 3,060,000 (Fouchet)—positive.

1/4/23: Hbg., 65 per cent.; red blood-cells, 3,000,000 (Fouchet)—positive.

1/16/23: Hbg., 60 per cent.; red blood-cells, 3,780,000 (Fouchet)—negative.

At the time of the last examination the clinical improvement was marked. Patient was able to walk about, and in accord with that the red blood-corpuscle count was markedly higher, and consequently blood destruction was less and bile-pigment in serum decreased, *i. e.*, Fouchet was negative. She returned with a marked exacerbation, and the following findings:

3/20/23: Hbg., 25 per cent.; red blood-cells, 1,235,000 (Fouchet)—double plus.

That is, with increased blood destruction and lower blood-count the Fouchet increased markedly in intensity.

Another case, L. E. On entrance:

3/8/23: Hbg., 45 per cent.; red blood-cells, 2,160,000 (Fouchet)—negative.

3/20/23: Hbg., 38 per cent.; red blood-cells, 2,150,000 (Fouchet)—double plus.

3/26/23: Hbg., 40 per cent.; red blood-cells, 2,150,000 (Fouchet)—positive.

Clinically this patient lost in strength far more seriously than is indicated by the change shown above in hemoglobin or red-cell count, rather more in accord with the change in Fouchet, *i. e.*, blood destruction may precede a drop in red-cell count. Not enough cases of severe secondary anemia have been examined to make sure that the test is consistently negative in them, but if so it may prove to be of diagnostic help in doubtful cases, especially as it is usually negative in carcinoma of the stomach except those directly involving the liver. It is of equal prognostic value with hemoglobin determinations and red-cell counts in determining remissions in pernicious anemia.

To sum up: Jaundice of both obstructive and dynamic types may be latent from the beginning to the end of the disease, and can then be determined only by bilirubin determinations in the blood-serum. This method of blood examination has definite diagnostic value in certain cases of acute catarrhal jaundice and cholecystitis.

CLINIC OF DR. RALPH C. HAMILL

CHILDREN'S MEMORIAL HOSPITAL

INFANTILE CEREBRAL PALSIES

INFANTILE cerebral palsies are obviously paralyses or disturbances of motion of cerebral origin in infants. They are characterized by associated involuntary movements, rigidities, convulsions, and mental deficiencies. One or all of these associated symptoms may be present.

Infantile cerebral paralyses differ from adult paralyses in certain particulars. In the matter of etiology infantile cerebral paralyses have two factors which obviously are not present in the adult, namely, the period of intra-uterine life and the trauma of birth. We can return to these factors and discuss them more at length later on.

Relatively speaking, acute inflammation plays a much more important rôle in children than it does in adults. Until the present epidemic, or the epidemic of the recent past, encephalitis or acute inflammatory disease of the brain played a rather small rôle in the causation of disturbances of movement in the adult. In children we know that influenza, whooping-cough, measles, and scarlet fever are followed all too frequently by disturbances of motion, poor mentality or epilepsy, indicative of inflammatory disease of the brain.

In the matter of symptomatology infantile cerebral paralyses differ from adult paralyses in four essential points:

1. The proportion of diplegias and paraplegias is much greater in infants than in adults.

2. Involuntary movements are seen as the result of brain disturbances much more frequently in infants than in adults.

3. Quite a large proportion of children whose brains have

been affected before birth, at birth, or in very early infancy have convulsive attacks. It is not improbable that there is some relation between the frequency of epilepsy in these cases and the involuntary movements of choreiform and athetoid nature.

4. It may be readily understood that damage to the brain of an infant will frequently be followed by deficiencies in the action of this brain in its highest respect, namely, mental activity. The proportion of mentally deficient children who have had some form of brain disturbance resulting in infantile cerebral paralysis is very high.

Infantile cerebral paralysis differs from adult also in the matter of pathologic basis. The factor of agenesis naturally is not found in adult cases, but it is quite possible that it plays a rôle in the paralytic and idiot. When a fetal brain is affected by infection, by traumatism in the mother's uterus, or even perhaps by emotion of the mother, it may show a very different picture after five or six years of extra-uterine life. Instead of showing definite scars such as would result from a disease affecting an adult brain the infantile brain may merely show a failure of development. The gyri may be less complex or smaller in size, the cell content of the gyri may be diminished or disarranged, and there may be an appreciable difference in size between the two hemispheres.

In adults when a paralysis is due to a hemorrhage it usually is in the structures of the base. We have all been brought up on the idea of the artery of cerebral hemorrhage, namely, the lenticulostriate branch of the internal carotid, whereas in infants the usual place of hemorrhage is on the convexity.

The brains of patients having had infantile cerebral paralysis show much greater destructive lesions than are ever found in adult brains. We need only think of the brains showing porencephaly, large defects in brain structure which may involve up to one-fourth of the hemisphere, extending from the convexity into the ventricle.

To return to the etiology: Since the condition, infantile cerebral paralysis, is not a specific disease, in fact, is quite the

opposite, it is to be expected that the etiology will be anything but specific. We may start off with one fixed condition, namely, infantile cerebral paralysis can be caused by anything that can injure the brain of the child before or at birth, or in the first decade of life. Of these things must first be considered failure of growth due either to inherent weakness of the germ plasm or to some abnormal direction of growth. This is mentioned first not because of its relative importance, but rather because it naturally occurs first to our minds when we are trying to consider the problem from its various aspects. When we have said that the condition is due to inherited defect or weakness we have said very little; however it is true that in certain families apparently not infected with syphilis a number of children will be found suffering with various forms of brain disease, various combinations of motor and mental disturbances. It has seemed in some of these families as though failure of development was the most logical and satisfactory explanation. In short, failure of germ plasm to develop into normally functioning brain merely through weakness in growth is perhaps a cause of infantile cerebral paralysis, but not an important one.

The rôle of trauma is still more or less of a mystery. Trauma to the child *in utero* seems the only explanation in some cases, yet it is not very acceptable when the trauma is relatively slight. There doubtless are cases in which the traumatic force is exerted directly on the fetal head, catching it against the rigid posterior wall of the maternal abdomen or pelvis.

Trauma at birth is universal. There is considerable molding of the skull that is inevitable in its passage through the birth canal, sliding of the upper edges of the parietal bones over each other with danger to the veins as they enter the longitudinal sinuses. There is also marked and prolonged stasis in the veins due to interference with return of flow. This molding is more marked in primipara with small pelves and powerful expulsive muscles. There is also the trauma of the forceps. This is a moot point. The obstetricians seem inclined to blame the conditions that require the assistance of the forceps rather than the direct trauma of the forceps themselves. This

point of view may be correct when the forceps are in the hands of a trained man, but forceps certainly are used by men not well trained, men who do not realize the dangers to the fetal head. The factor of venous stasis may play a more important rôle in cases of breech presentation where the after-coming head is subjected to additional danger. The head with its veins overdistended has to undergo the usual, or perhaps an unusual, amount of molding. Hemorrhage is induced by asphyxia and so the molding may be so much the more dangerous when pressure shuts off the flow in the umbilical cord.

Trauma to the infantile head certainly causes hemorrhage in some cases quite independently of other factors, namely, weakness of the blood-vessels, and the hemorrhage is usually over the convexity. Also trauma to a portion of the body seems to predispose to an involvement of that portion by inflammation.

Behind many of these cases of trauma, as well as independently thereof, is the matter of inflammation. It is possible that the brain of the unborn child may become the seat of an inflammation of maternal origin. That inflammation may have been very slight in its effect on the mother and have done its damage and entirely subsided by the time of birth. Also, inherited or congenital syphilis may have affected the brain of the unborn child without being apparent elsewhere. In the years of childhood inflammation seems to show a great fondness for the brain. This predilection may be more apparent than real. It may be that inflammation may destroy portions of other viscera and the function of the viscera be carried on by other portions thereof, whereas there are few areas of the brain that can be spared, and the scars of such destruction act upon the remainder of the brain in a manner foreign to the other organs. At any rate, any of the acute inflammatory diseases of childhood may start in with, or show at some time in their course, sonnolence, rigid neck, and convulsions—signs of involvement of the brain. Influenza, measles, scarlet fever, whooping-cough are especially apt to be so complicated; they appear very frequently in the history of the child who is epileptic, paralytic, or an imbecile.

Inherited syphilis must always be ruled out. The Wassermann reaction may be negative in the child and in one parent and yet positive in the other, or the Wassermann reaction may be positive all the way through. Then the solution is simple. There are other signs of syphilis in the infant that must be looked for—the square, bulging forehead, the short, broken, feeble nails, the late and faulty eruption of teeth that may show pitting, notching, and chisel shapes. There are also the snuffles, the body rashes, and lymphadenitis to be looked for.

In spite of the variety of causative factors referred to we are often at a loss in our search for the cause.

Symptomatology.—Cases of infantile cerebral paralysis present themselves as hemiplegias, diplegias, and paraplegias. There are also cases in which the various abnormalities of movement—chorea, athetosis, associated movements—appear without true paralysis. There are also cases of feeble-mindedness and epilepsy which are due to destructive lesions of the fetal or infantile brains.

The cases in which the paralysis or motor disability is more marked on one side than on the other are very numerous. There are true hemiplegic cases in which the hemiplegic weakness is quite strictly confined to one side of the body. In these cases there are usually some signs from the other side of the body, an increase in the deep reflexes, even, as is quite frequent, considerable spasm in the muscles of the opposite leg.

HEMIPLEGIA, PROBABLY BIRTH INJURY AND HEMORRHAGE

Case I.—Robert N., aged three years and eight months. Second pregnancy; the gestation period was normal except that there was much vomiting during the first half. Labor was only two hours long and no instruments were used. A few hours after birth the child was observed to be cyanosed, which the doctor in attendance attributed to congenital leakage of the heart. He was not able to nurse until the third day. The first few months were uneventful, but at the age of six months the mother noticed that he did not use the left arm. He sat up at one year and walked at two years. There was no acute

illness during infancy. At about one year of age one convulsion is said to have occurred. Apparently there was no delay in talking. He can point accurately to different parts of the body when they are named. His complexion is sallow. He walks with a typical infantile hemiplegic gait (Fig. 67). The left leg is turned in, slightly flexed at the knee; the foot is in extension

Fig. 67.—Infantile hemiplegia.

and the boy walks on his toes. The left arm is carried flexed at the elbow, and the wrist slightly abducted and rotated inward. There seems to be no failure of growth of the left extremities. The left face shows a very slight tendency toward spasm and there is a slight tendency to convulsive laughter. The eye and tongue movements are normal; the teeth are good and the

pharynx is apparently normal. There is well-marked resistance to passive movements in the left leg and also in the left elbow; this is less marked in the shoulder and wrist. The left leg and foot are a trifle cooler than the right. He shows only the faintest trace of associated movements in the right hand when attempting

Fig. 68.—Athetoid posture of left hand in reaching.

voluntary movements with the left. There are very well-marked associated movements in the right foot when he is asked to move the left. Voluntary movements, such as reaching with the left arm, show good control in the shoulder, but with an athetoid accompaniment in the fingers. With this reaching there are associated movements in the right arm (Fig. 68).

The deep reflexes in the left arm are exaggerated; in the right they are perfectly normal. The abdominal reflex is brisk; the cremasteric is brisk, with contralateral reaction on both sides. The right testis is in the canal. The plantar reflexes to gentle stroking are indefinite; to harder stroking there is a Babinski sign on the left side, with a contralateral on the right. The left ankle-jerk is greater than the right; the knee-jerks are exaggerated, but equal. The adductor jerks are brisk. There is an abnormal flaccidity of the left toes and fingers. His mother states that he has been tidy in his habits for some time.

Comment.—This is a case of hemiplegia, probably due to cerebral hemorrhage, occasioned by the trauma of birth. The fetal vessels may have been predisposed to rupture by the maternal vomiting throughout the first half of pregnancy. Though the cyanosis was ascribed by the doctor to congenital heart trouble, the absence of any sign of heart trouble now and the lack of other history of etiologic importance leads me to believe that the cyanosis seen a few hours after birth was due to respiratory difficulty induced by intracranial hemorrhage.

The symptoms characteristic of infantile cerebral paralysis are the convulsion at one year and the physical signs now present, namely, the spasmodic or hypertonic appearance of the paralyzed side of the face and the convulsive type of laughter; also the movements of the right foot and hand when attempt is made to move the contralateral members; also the abnormal flaccidity of the fingers of the paralyzed hand, and perhaps we must include contralateral reflexes and the undescended right testicle. It is difficult to determine the degree of mental progress in a case of this kind. The spasmodic emotion is bound to give an idea of feeble-mindedness; perhaps that is as it should be. If intellectual processes are not sufficiently strong to control emotional outbursts there is that failure of self-control which is prima facia evidence of lack of development.

CEREBRAL DIPLEGIA

The cases that are more typically infantile, that differ from any motor disturbance seen in adults, are the diplegias and

cerebral paraplegias. In the diplegias it is often difficult to determine whether the paralysis or the spasm is more marked; the paralysis may be due to the spasm. Involuntary movements may interfere with normal function to such a degree that purposeful movement practically does not exist.

Diplegia, a Twin; Birth Injury.—Case II.—This brain that I am showing you (Fig. 69) is that of a man who died at thirty, a case of diplegia with involuntary movements and mental

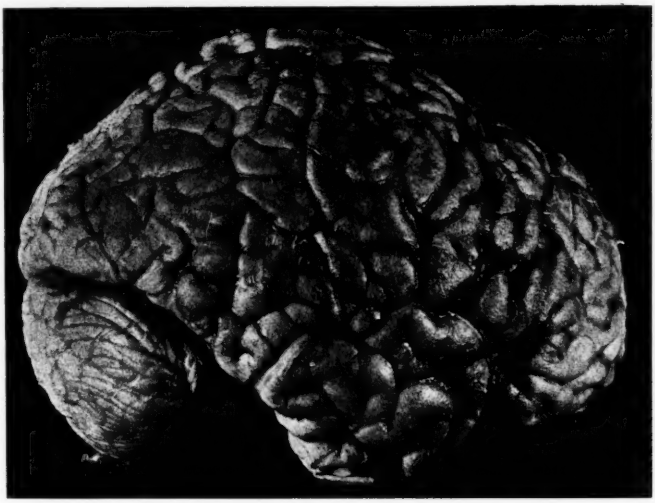

Fig. 69.—Atrophy of frontal gyri.

enfeeblement. When looked at from the side the smallness of the frontal lobe is obvious; in the frontal and lateral views this lack of proportion is explained by the atrophy of the gyri of the frontal lobe. Here at the posterior end of the inferior frontal and in the anterior portion of the middle frontal this atrophy is clearly evident (Figs. 69, 70). The pia is thickened and edematous over the frontal lobes. On cut section the ventricles are distinctly enlarged. There is no gross lesion of the

structures at the base of the brain. The pyramidal tracts in the cord stained with Weigert-Pal were normal.

The patient was the first to be delivered of a pair of twins. His was an instrumental delivery. The second twin was born without instruments. The patient was a blue baby; it required two hours' work to bring him to. He was considered normal for the first few months. Then, when his twin brother began to make purposeful movements, it was noticed that the patient

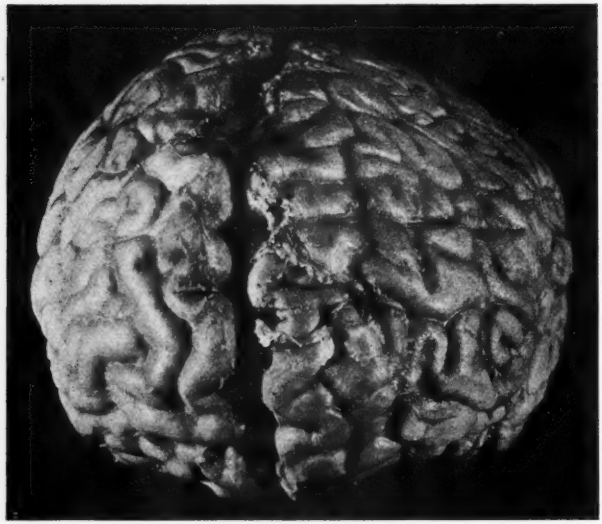

Fig. 70.—Frontal view.

would merely throw back his head and go into a sort of spasm. He was nervous and did not sleep well. This photograph, taken when the twins were about nine months old (Fig. 71), shows but little. The startled expression on the baby to the left indicates the patient. The position of his right hand is probably determined by the same forces which act to produce the distortion seen in this other picture taken when the patient was twenty-five (Fig. 72).

At the age of eighteen months he had his first convulsion. At

about seven years he tried hard to walk, but he was quite unable to overcome the spasm in his legs; they became quite rigid and without support he would fall like a log. Attempts at speech merely threw him into convulsive movements of the trunk and limbs and his face would be contorted with grimaces. The grimacing would occasionally terminate in a sort of explosive laugh. One got the impression that the ideas bearing a minimum of emotion were yet able to stimulate emotional centers and produce emotional expressions. His mother considered him the brighter of the two boys, "because his memory was so good."

Fig. 71.

Anything that occurred in the house or in the family circle was remembered with great accuracy of detail by the paralyzed child. Because of the failure of development of his legs he spent his entire time either in the house or in its immediate neighborhood. The happenings in the home were few and far between, they were not diluted by great numbers, and most of them had some close relationship to his comfort or pleasure. Hence his memory could reproduce them distinctly and in detail, unclouded by memories of thousands of happenings encountered by the freer moving brother.

Breathing was distinctly shallow and swallowing was largely

a matter of chance; that is, the liquid or solid might go down the esophagus or it might try to come out of his nose or get into his larynx. He was brought from his home, where he had spent his sheltered life, to the hospital and immediately began to fail. This move was made because death in the family made his further care at home impossible. In the first few days at the hospital he choked much more often than he had at home.

Fig. 72.

His breathing seemed more clumsy: there was a sound as of the soft palate interfering with expiration. It seemed as though terror had interfered with his vital reactions.

No movements except those of the eyes could be made upon request, and even those could not be counted on surely. His eyes would follow the moving finger a short way, then the involuntary movements would jerk his head, or even his eyes

would seem to be incontinently jerked away. Requests for movements of the face, tongue, or upper extremities produced an explosion of abnormal exaggerations in which the face, neck, arms, and legs all took part (Fig. 72). Ocular reflexes were normal, deep reflexes in the arms were exaggerated; the abdominals and cremasterics could not be elicited; reflexes in the legs were absent. So far as could be determined sensation was normal.

The head was smaller than normal, the trunk was fairly well developed, the muscles of the arms stood out, but were not well developed. The legs were very short; apparently they were about two-thirds the length expected with such a trunk and arms. Their muscular development was much inferior to that of the arms and trunk.

Comment.—This appeared to be a case of birth trauma; the trauma of an instrumental delivery which caused hemorrhage over the frontal regions. As a consequence of this hemorrhage there was a fairly uniform, superficial destruction of that part of the brain which is concerned with ideation and its relation to motion. Clinically it was a case of diplegia with very marked involuntary movements and feeble-mindedness. There was one convulsion. A very interesting side was that of the weakness of the fundamental reflexes of breathing and swallowing. Apparently he was getting on fairly well at home, but when he was brought into strange surroundings his fear reactions were stirred up to such a pitch that they interfered with these reflexes. It seemed very evident to those observing him that he died because of this interference.

Diplegia; Probably Inherited Syphilis.—Case III.—Carter B., colored, is two years and nine months old. This was the first pregnancy; gestation was normal. Labor extended over twenty-five hours, it was a head presentation and no instruments were used. Birth was normal. He did not suck (a bottle) until a week after birth, and was fed with a spoon during this period. There is no history of convulsions. He did not sit up until he was two years and five months old and does not walk now. His

mother states that he said "papa" at one year, and that he can now say everything, but the father says that only the mother can understand what he says. His voice, as you observe, is very high pitched, feeble, and his speech is syllabic. His mother says he usually indicates to her when he wishes to urinate. His gait, as he walks on tiptoe when supported by his father, is typical; it is spastic, cross-legged, the feet interfering because

Fig. 73.—Cross-legged posture of cerebral paraplegia.

of adductor spasm (Fig. 73). The adductor spasm is also well marked when he is held up with the hands about the body underneath the arms.

The contralateral eye turns in when he looks toward either side; when looking straight ahead either the left or right eye may turn in (Fig. 73). Winking is normal. The pupils are normal. The eyeballs are constantly affected by a very fine

nystagmus which is especially marked in all extreme positions. The emotional expression is symmetric; there is no facial paresis.

Grasping movements with the left hand show clumsiness and overaction; these are of an athetoid nature. There is a bilateral Babinski especially marked on the left side. The knee-jerks are exaggerated; the ankle-jerks are brisk, but the feet are held in such extension as to make tests worth but little. The abdominal reflex on the left side is absent; on the right greatly diminished. The deep reflexes of the arms are all greatly exaggerated.

Carter's attention can be gained, but not held; one might say that his interest can be engaged, but it is not self-sustaining; when once started it is not self-perpetuated, it fails as if from weakness. In a boy of this age it may be only from signs of this kind that mental defect can be determined.

Comment.—This is a case of infantile cerebral paralysis of the paraplegic type, with some involvement of the arms also. There is spasm more than paralysis. He is probably feeble-minded, though at this early age evidences thereof are very difficult to elicit.

Inherited syphilis is probably to blame for this condition. The Wassermann reaction is negative both in the blood and the spinal fluid, as is the Lange in the latter. It is negative also in the mother's blood, but the father's blood gives a ++++ positive.

The fact that Carter did not suck until he was a week old suggests that there was some defect at birth. No instruments were used and nothing occurred at birth to suggest birth injury or hemorrhage; hence, we are forced to think of some disease affecting the fetal organism. Gestation was normal, but syphilis may have been present and unsuspected. Negative Wassermann reactions are not uncommon in children damaged by inherited lues. If the disease has been active, scarred the central nervous system, and its activity died out one can readily understand that the reactions may be negative. At any rate, Carter is to receive antiluetic treatment.

Feeble-minded; Slight Hemiplegia; Encephalitis at Fifteen Months.—Case IV.—Lena is two years and two months old. The gestation period and birth were normal and she was all right until she was one year and three months old. At this age, after an apparently normal day, she went into what the mother described as "*krampfe*," in which condition she remained unconscious for twenty-four hours. At frequent intervals during the following week there were periods of convulsions and she was thought to have had fever during the first half of the week. Before this attack the child spoke and walked normally, but since that time she has not spoken, and for eight months after the acute illness she did not walk. She has cried a great deal since the illness.

During the examination the child alternately masturbates and sucks her fingers. During the time she is engaged in the former she constantly keeps up a plaintive crying. She is pot-bellied and obviously rachitic. She walks fairly well, though careful observation shows a little favoring of the right side. Right stepping is shorter than left. She has had no convulsions since the onset. The plantar reflexes on both sides are suggestive of the Babinski sign. The ankle- and knee-jerks are normal; the abdominal reflexes are diminished. The wrist-jerks are normal.

It is to be observed that she always uses her left hand both to suck and for reaching; the mother says that she does most things with her left hand. When it is possible to get her to reach with her right hand there is considerable clumsiness and over-action of the athetoid type.

It is almost impossible to get the child's attention. She sees and hears, but either fails entirely to understand or cannot be appealed to by things that ordinarily interest children of her age. A child of two should be talking with a fair command of words to express her wants and interests.

Comment.—This case is one of feeble-mindedness with slight motor disturbance of the hemiplegia type probably due to an acute inflammatory disease of the brain at fifteen months. The motor disturbance is seen chiefly in the athetoid or clumsy

action of the right hand and in the slight weakness in walking of the right leg. When the child attempts to grasp my finger or percussion hammer with the right hand her fingers are spread and hyperextended, and then the fingers and thumb rather slowly fold around the object. It is too early to fortell the outcome so far as speech is concerned. It probably will return, but will never be normal; it may be slower in returning because the preponderance of the lesion is in the left hemisphere.

Symptomatology.—The symptoms of infantile cerebral paralysis can be considered under the general headings of:

1. Disturbances of purposeful movement.
2. Epilepsy.
3. Feeble-mindedness.

1. If in retrospect we try to generalize concerning the disturbances of motion in infantile cerebral paralysis we have to realize that paralysis is not a satisfactory word. Occasionally there is too much movement rather than a loss thereof. The associated movements, the chorea, and the athetosis, these are not samples of what we ordinarily have in mind when we speak of paralysis. Yet if we think of paralysis as something interfering with purposeful movement, then these disturbances of infantile cerebral palsies all belong. Perhaps we can best think of the disturbances as due to interference with voluntary control of movement, such as we are all busily developing during our infancy and childhood. If the brain is normal we gradually can and do develop the ability to specialize infinitely in the variety, exactness, and strength of movement; if the brain is damaged that development is impeded.

There are cases of infantile cerebral paralysis with merely a clumsiness of motion which in some instances may determine a left-handedness through interference with development of accuracy in the right hand. I have seen one shining example of such a case. The doctor in charge of the military camp hospital was going to send this man in replacement battalion to France in the fall of 1917. On cursory inspection there seemed nothing against such a course, but my attention was attracted to him by an internal strabismus of the left eye, a condition known to

have existed from earliest youth. On careful inspection the left face was seen to be smaller than the right. This combination looked as though the left sixth and seventh nerves might have been involved in some pathologic process; in other words, the strabismus was not a matter of a misshapen eyeball. The soldier was taken into the hospital and stripped. The right arm and leg were found to be 2 or 3 cm. smaller than the left. He was left-handed. He hopped very clumsily on the right leg, quite well on the left. He was a very sturdy young man, and even with these handicaps the Colonel was for sending him to France. I forgot to mention that he rated a mental age of ten. Fortunately for the A. E. F. he made homosexual advances to other men in the barracks, whereupon he was promptly let out on an S. C. D. A questionnaire sent to his family brought the information that he was born with great difficulty, that he was a blue baby, that twenty-four hours after birth he had a convulsion, and from then on his left eye turned in. A hemorrhage must have occurred that damaged the pons or medulla in the region of the sixth and seventh nuclei on the left side and involved the pyramidal tract above its decussation. This led to the strabismus, the smaller left face, and the clumsy, smaller right arm and leg. To the superficial observer this was merely a retarded left-handed boy from the mountains of Kentucky.

Associated movements are quite common in infantile cerebral paralysis. When a patient is making an intense effort with the paralyzed member there may be movements in the symmetric member obviously of a nature asked for in the paralyzed. For example, as the hemiplegic child tries to make a fist with the choreic, spastic hand, the normal hand begins to gather into a fist. As she tries to move her paralyzed toes she cannot keep the normal toes still.

Choreiform and athetoid movements are very common in infantile cerebral paralysis. They may be of sufficient degree to dominate the picture and quite overshadow the paralysis, as can be seen in the picture of the boy in the chair (Fig. 72). In his case the involuntary movements amounted to disabling gyrating spasms. All four limbs and the muscles of the face

would go into and change out of all sorts of extreme contorted postures, sometimes slowly and sometimes very rapidly. This exhibition might be set up by speaking to him, making a sudden noise, or even apparently by the arising of some thought in him that called for expression. The same extreme of motion might be suggested by the athetoid posture assumed by the fingers of the left arm in Case I, but the abnormality was confined to the fingers and occurred only momentarily during the course of an otherwise fairly regular and purposeful motion. In both Case I and Case II there were abnormal emotional reactions. The motor expressions were apparently set off much more easily than they should have been. Convulsive laughing is rather common in infantile cerebral paralysis. It is as though the intellect was insufficiently developed to maintain normal control of emotion.

2. Epilepsy or convulsive seizures are very common in infantile cerebral paralysis. The history may show spasms at birth—one, two, or more—and then nothing more indicative of the seriousness of the trouble until at five or six months a failure to make purposeful movements is noted. Or it may be that nothing is noted until fifteen or eighteen months and the child is too unsteady to walk, or does not begin to speak. Such a history as this is all too common: birth normal, life uneventful until twenty-four months, then a series of convulsions, six or eight in number, thought to have been caused by something the boy had eaten; then apparent normality until the age of thirteen, when he screamed, became violent, and had a convulsion; thereafter the convulsions recurred, though never very severe, every month to every week.

When the convulsions are very frequent and severe they may so dominate the picture that they are to be considered as the cause of the feeble-mindedness and motor disturbances. There is no question but that cases have been observed in which after months or years of more or less frequent convulsive attacks a motor disturbance has appeared. The convulsions may occur at four to six months' intervals, then suddenly become quite frequent, two or three a day, and then every day or every week,

or they may show a surprising degree of regularity. They may occur only in the daytime, but are more apt to occur both day and night.

It may be very difficult to differentiate the temper-breath-holding attacks of infants from epilepsy. In the former condition there is the violent outburst of temper, with screaming, kicking and fighting, then rigidity in the expiratory phase, turning blue, relaxing, and the resumption of breathing. There is none of the typical clonic phase of epilepsy. It may be still more difficult to distinguish the spasmophilic in the very young, and it may require calcium feeding to be certain, though the Chvostek and Trousseau signs may help. Also, feeble-mindedness and any of the disturbances of motion common to infantile cerebral paralysis would indicate epilepsy.

3. Feeble-mindedness: The brain is the organ of thought. Just how much of it is necessary for thinking or how much can be spared we do not know. It has often seemed to me that no part can be spared and normal (for that individual) thinking go on. Thinking is such a complex matter and depends so much upon the establishment and maintenance of all possible associations that it is difficult to believe that any considerable portion of the brain can cease acting and thinking go on undisturbed. Of course, the word "thinking" is used very loosely and but few know what they mean thereby. The mother of Case II thought him "brighter" than the other twin simply because of his memory. She took no account of his failure of wide interest, failure of appreciation of his position in the world; his inability to read or write naturally could be excused by her and blamed on his motor difficulties.

There may be some of these cases of infantile cerebral paralysis who develop into normal, thinking adults, but they certainly make up a small percentage. The deficiency may show itself merely as an inability to realize or appreciate an audience. By that I mean that children willingly say anything and expect to be believed, but as they grow older there develops a realization of their own limitations and of the auditor's ability to think around what he hears. The child develops an ability to project

himself into the minds of others. But children who lack this ability usually also find great difficulty in getting on in school; two or three years are spent in the first grade because of lack of application, which bespeaks a lack of interest. The defect may be so great that school is never attempted, speech is too indistinct, attention too difficult to fix, conduct too uncontrolled, and irresponsibility too great.

There are also the idiots who drool, wet and soil themselves, who can be taught almost nothing. Perhaps they know only the sound of the voice of someone who feeds them.

In other words, there are all degrees of mental defect, from the barely retarded to the unthinking idiot, depending upon the amount of brain destruction and the age at which it occurs. It may be asociated with very minor motor difficulty or may be found in all grades in the completely helpless.

Therapy.—Treatment in this condition is not an interesting or encouraging subject to discuss. Naturally, but little if anything can be done to the brain. It is scarred and there is no use in trying to remove the scar.

Preventive medicine is most important. If there is any suspicion of syphilis the mother must be given intensive treatment. It would be better to have an abortion or a dead baby than one suffering from paralysis, epilepsy, and feeble-mindedness.

Birth procedure must be carefully watched. Too long duration of cyanosis is dangerous. The forceps must be used, but should be used only under skilful guidance.

If the newborn child fails to breath quickly or has spasms, or shows weakness of the sucking reflex, or its deep reflexes are unequal, opening of the skull must be seriously considered.

The orthopedist can do a good deal to increase the function of the patient in certain cases. In the first place, the patient must have a fair degree of intelligence; he must be teachable, and when given an idea be able to keep to it; in the first line of treatment are passive movements and massage, by which means spasm may be sufficiently overcome to allow of a fair degree of action by antagonists. If the spasm and contracture is too great to overcome by these means, tenotomy may allow the an-

tagonists to work. This type of procedure is most useful in the legs, where tenotomy of Achilles' tendons, the hamstrings, and the adductors may change a non-functioning pair of legs to fairly satisfactory supports. The after-care is most important. The limb must be so immobilized as to give the antagonists a chance to contract. As soon as possible passive and then active movements must be begun to allow the antagonists to develop maximum power.

There is no curative treatment of the motor restlessness and inco-ordination. Bromids may be required to combat periods of excessive nervousness. Also, bromids or luminal are useful in keeping at a minimum the convulsive episodes.

Feeble-mindedness really demands institutional care. The presence of an imbecile in a group of children is mutually unfortunate.

It is quite possible that many cases of infantile cerebral paralysis are never recognized; individuals with slightly retarded mentalities, also so-called idiopathic epilepsies. In the case of the former the degree of mental retardation may unfit them for brain work, but not interfere with the making of a good common laborer. Education along manual lines is indicated in the cases where the mind is more harmed than the muscles. Much can be taught the cases that seem quite hopeless. They are taught to participate quite extensively in their own care. They may be able to speak fairly well at eight, when at four speech seemed almost impossible. With patience an athetoid hand may be gotten under fairly good control. But any such teaching is usually wasted effort. Except the few, these patients can at best be laborers and not very productive ones at that, and the expense and patience put on one of these cases would be immensely more rewarded by the normal brother or sister.

CLINIC OF DRS. FREDERICK TICE AND
VINCENT J. O'CONOR

WASHINGTON BOULEVARD HOSPITAL

DISEASE OF THE UPPER URINARY TRACT WITHOUT REFERABLE SYMPTOMS

IN presenting the following cases we wish to emphasize the value of resorting to routine urologic study for the completion of an accurate diagnosis in the more obscure abdominal complaints.

It is needless to state that individuals with pronounced urinary symptoms, such as frequency, dysuria, hematuria, or pyuria, will demand a careful investigation of the urinary organs. On the other hand, many patients in whom lesions of the urinary tract exist have no typical symptoms referable to the functions of these organs. Not only are symptoms suggesting urinary disease absent, but in these cases the urine is frequently normal both chemically and microscopically.

The absence of symptoms directly pertinent to the urinary tract and the lack of urine abnormality too often lead the diagnostician to exclude the possibility of pathology in these organs. As a result many of these individuals are subjected to exploratory laparotomy, with subsequent removal of appendix, gall-bladder, or uterine adnexa in an effort to relieve these vague abdominal complaints.

The type of lesion causing these conditions is practically always an obstruction to proper urinary drainage, and can only be discovered by visualization through the cystoscope or by outlining the urinary tract by special methods in conjunction with the *x*-ray.

Case I.—Clara C., an American housewife, aged twenty-seven years, presented herself on August 21, 1922, complaining

of severe pain in the left lower abdomen and left costovertebral region.

Family history unimportant.

The patient had none of the usual diseases of childhood and, other than present complaint, had always been well. Habits and general health normal. No accidents or body blows.

Complaint on admission was of five years' duration and consisted of recurring attacks of severe pain in the left lower abdomen. The area of pain distribution had always been somewhat vague, at times extending to the costal margin in front and at other times involving the upper left lumbodorsal region. While severe, the pain had never been "colicky" and had never radiated into the left groin, perineum, or thigh. The severity of the dull ache or "pressure soreness" often compelled her to lie down for several hours. Deep pressure over the lower abdomen often assisted in relieving the pain. The pain was never associated with, or affected by, urination or defecation. No dysuria, frequency, pyuria, or hematuria had ever been noted. Hot douches or baths and morphin by mouth were the only methods at all successful in relieving the pain.

The patient had no gastro-intestinal symptoms, was not constipated, and had had normal menses since the age of fourteen.

In June, 1920 she was subjected to laparotomy, with resultant removal of appendix, left ovary and fallopian tube, and fixation of the uterus. These procedures brought no relief.

The patient stated that several of her medical advisers had considered the possibility of urinary tract disease, but since roentgenograms of bladder, kidney, and pelvis were repeatedly negative and frequent urinalyses were normal, this conjecture had been abandoned.

For the past six months the pain had been less severe in the left abdomen, but a constant backache had persisted. She had never felt rested, and had lost all physical ambition.

Physical examination is negative in every respect except for tenderness when deep pressure is made over the left lower abdomen. There is also a slight costovertebral tenderness on

the left. There are no palpable masses or other areas of tenderness in the abdomen. Neither kidney can be palpated. Bimanual examination shows no abnormality in the pelvis. Blood-picture normal. Leukocytes 8000. Temperature, pulse, and respiration normal. Plain roentgenograms show normal kidney outlines and no shadows which could be interpreted as calculi. Two stool examinations gave no abnormal findings. The urine was carefully examined in single specimens and in twenty-four-hour amounts, and was repeatedly normal.

A tentative diagnosis of obstruction in the course of the left ureter was made and cystoscopy advised.

Cystoscopy.—The urethra was normal. Bladder capacity 300 c.c. The cystoscopic picture at first was very confusing, but after distending the bladder to its full capacity the pathology was clear. The floor and lateral walls of the bladder were normal and there was no congestion or edema. The right ureteral orifice was normal, with clear efflux. The entire left half of the trigone was obscured by a grayish-white mass which projected downward from the region where the left ureteral opening would normally be seen. On closer study the wall of this pouch presents vermiform movements with feeble peristaltic waves toward the bladder neck. It is then clear that the condition is due to a cyst-like dilatation of that portion of the ureter which runs through the bladder wall. This, then, is a ureterovesical cyst. The orifice of the left ureter could not be found.

The diagnosis thus far is clear. The patient's pain is due to the obstruction in the left ureter at this point, causing back pressure and distention of the left ureteral wall and, probably, renal pelvis. The next thing to determine is whether the ureteral dilatation has resulted in irreparable kidney tissue destruction. but this is impossible because the left ureter cannot be catheterized. The protruding cyst wall, however, apparently constitutes the obstruction, and if this is removed the integrity of the ureteral opening will be established.

On September 2, 1922 the patient was again cystoscoped under scopolamin and morphin anesthesia, and by means of a large pointed electrode the entire obstructing cyst wall was

destroyed by the Oudin current. Twenty-four hours after this procedure the patient passed a large quantity of coagulated débris through the urethra, following which there was no discomfort.

Fig. 74.—Dilatation of ureter above left ureterovesical cyst. Picture made after destruction of obstructing cyst wall.

She stated the next day that she was absolutely free from soreness on the left side, and was able to make pressure in this region without the slightest discomfort.

Three weeks after the destruction of the cyst wall the patient was again cystoscoped. During the interim she had been entirely free from pain or discomfort.

A catheter could be easily passed up the left ureter for a distance of 30 cm.

Kidney Function Tests.—Phenolsulphonephthalein, 1 c.c., injected intravenously appeared: right three minutes; left, four minutes. Quantity excreted in thirty minutes: right, 25 per cent.; left, 10 per cent.

A left ureteropyelogram was then made by injecting 15 per cent. sodium bromid. This showed a diffuse dilatation of the lower third of the ureter, with a gradual tapering up to a practically normal ureter and kidney pelvis above.

The fact that there is no blunting of the calices or dilatation of the pelvis indicated that this kidney would be restored to its normal function, since the obstruction had been removed. A ureteral bougie has been passed through the left orifice at monthly intervals four times to insure the persistence of its patency.

The patient states that she is absolutely well and has no pain of any kind.

A thirty-minute phenolsulphonephthalein test yesterday showed an excretion of 20 per cent. of the dye from the left kidney.

This case is instructive because it emphasizes the possibility of urinary tract obstruction as the cause of indefinite distress, even when subjective and objective symptoms of urinary disease are lacking.

The rapid reparative power of kidney excretion after the removal of incomplete obstruction in the lower ureter is also demonstrated.

Case II.—Mildred T., American, schoolteacher, aged twenty-six years, presented herself November 2, 1922, complaining of distress in the right abdomen. An appendectomy four years previously failed to bring relief. Before coming here she had been in bed for two weeks under treatment for "colitis." As long as she remained quietly in bed she experienced partial relief from acute pain, but a dull throbbing sensation was frequently persistent. The pain localized along the right costal margin

and sometimes extended into the right lower quadrant, so that the entire right abdomen was "sore" on deep pressure. There was no pain in the back or flank. The pain was never colicky, and did not radiate into the groin or genitalia. The family and past histories were unimportant.

General physical examination was essentially negative. A sense of pain existed only on bimanual palpation directed deeply into the right upper quadrant. Neither kidney was palpable. No tumor masses were felt in the abdomen. Vaginal examination was not made because of an unruptured hymen. Rectal examination showed uterus and adnexa to be normal in size and position.

Complete blood-count was normal. Blood Wassermann was negative. Urine: specific gravity, 1.014; acid, no albumin, no sugar, bile, indican, or blood-pigment. Sediment negative. Repeated urinalyses were normal.

Since this patient had previously had gastro-intestinal studies in another hospital, with stated negative fluoroscopic findings, it was decided that the most probable source of the pain existed in the right kidney.

Plain roentgengrams of the abdomen and pelvis showed no shadows that could be interpreted as calculi. The left kidney outline was normal. The right was obscured by gas in the bowel.

Two-hour phenolsulphonephthalein test showed an excretion of 65 per cent. Blood urea nitrogen was 15 mgm. per 100 c.c.

It is evident that no positive data was gained from physical examination, laboratory, or Roentgen studies.

Cystoscopy.—Bladder capacity 400 c.c. Bladder wall, trigone, and ureteral orifices were normal in every respect. Both ureters were catheterized for a distance of 30 cm.

From the right a rapid flow of clear watery fluid appeared. No dye appeared from this kidney for ten minutes after intravenous injection, and only 2 per cent. was eliminated in fifteen minutes. There was no albumin or any casts or pus-cells from the right kidney.

The urine from the left kidney was normal in every respect.

The dye appeared in two and a half minutes after intravenous injection, and 20 per cent. was excreted in the first fifteen minutes.

Ureteropyelogram on the left was entirely normal. On the right you can see the complete destruction of typical pelvic or calix outline. There is a hydronephrotic sac which has a capacity of from 8 to 10 ounces.

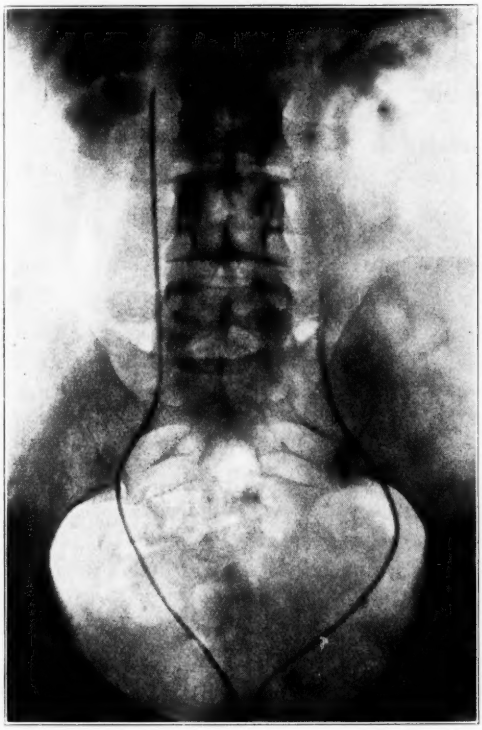

Fig. 75.—Right hydronephrosis shown by extensive destruction of renal pelvis and calix outline. Normal left ureteropyelogram.

A right nephrectomy was advised and accepted. The kidney was delivered through a right lumbar incision. A careful exploration of the region of the junction of ureter and pelvis revealed the underlying cause of the obstruction. At this point

an aberrant artery crossed the ureter to enter the kidney substance. It crossed just behind the ureter, and in such a way as to compress the ureter very much the same as a garden hose would be if thrown over the back fence.

In this case we have another instance of the value of urinary tract investigation in the event of a right hydronephrosis due to constriction of the ureter by an anomalous renal artery. The pain in this case should have indicated this study long before, especially in view of the negative findings in the intestinal tract and other body systems.

CLINIC OF DR. VINCENT J. O'CONOR

WASHINGTON BOULEVARD HOSPITAL

POSSIBILITIES OF ERROR IN THE DIAGNOSIS OF RENAL CALCULI

I WISH to discuss with you today the salient points involved in the completion of an accurate diagnosis of urinary calculus. In no field of medicine or surgery are the preoperative diagnostic methods so reliable in the detail of their findings as those we now apply to the localization or differentiation of shadow-casting bodies presumably in the urinary tract. The negative information that can be gained by these studies is equally important.

The common sources of error in arriving at an accurate diagnosis of calculi are as follows: misinterpretation of the clinical symptoms of disease, especially as to "types" of pain; failure to obtain *x*-rays of the possible "stone-bearing" areas of the body; in the event of *x*-rays being made, lack of appreciation of the relative values and proper interpretation of such shadows as may be found in the *x*-ray plate; ignorance on the part of the diagnostician in not appreciating that renal calculi may be present without either objective or subjective symptoms. In this regard we must remember that from 10 to 15 per cent. of all urinary calculi cannot be demonstrated by the most perfect plain roentgengram.

It should be well recognized that the classical or text-book description of renal colic, the so-called Dietl's crisis, of pain sharp and lancinating, referred from the lumbar region along the course of the ureter to groin, vulva, testicle or thigh, may hold true in one patient, while in another, who bears an identical lesion, this type of pain may be absent or diversified. It is not possible in the majority of instances to clearly exclude other

pathologic conditions because the location and character of pain in a given case are "absolutely typical." For example, an individual presenting what appears to be a typical gall-stone colic plus the presence of suspicious shadows in the radiogram, added to the fact that there have never been any urinary symptoms and that repeated urinalyses are normal, may still be suffering from right renal calculi. I will discuss such a case in a few moments.

The presence of shadows in a given radiogram indicates merely that there are present areas of varying degree of density. In most instances it is necessary to confirm both the location and character of these opacities before an absolute opinion regarding their significance can be given.

If the suspicious shadow is in the bladder region it can be confirmed or rejected by visual examination of this viscus. If it concerns the kidney or ureteral regions the passage of catheters impregnated with lead into the ureters and the subsequent injection into the kidney pelvis of a solution opaque to the *x*-ray will be necessary to obtain the proper data. To repeat, the information obtained from a simple *x*-ray examination of the urinary tract is of great importance, but only when confirmed by accessory diagnostic methods—ureteral catheterization and ureteropyelography.

Let us enumerate quickly the various shadow-casting bodies which we may be called upon to differentiate. In this problem, as in all others, it is well to bear in mind the list of possibilities, so that individual attention may be given to the exclusion of each.

Shadows that may be confused with renal calculi are: (1) Those due to material in the bowel. Among these should be mentioned fecal masses, enteroliths, fruit pits, and all opaque salts or shadow-casting pills, such as bismuth, barium, salol capsules, Blaud's pills, etc. It should be especially borne in mind that following gastro-intestinal studies with barium some of this residue may persist in small diverticulæ of the colon for quite some time. This has been especially confusing in some cases that I have seen where no preliminary plain *x*-rays had

been made prior to fluoroscopy. An enterolith lodged in a retrocecal appendix has twice necessitated my excluding it as a possible ureteral calculus.

(2) Gall-stones. (3) Calcified lymph-nodes, either from the lumbar or mesenteric chain. (4) Areas of calcification in the kidney substance due to tuberculosis or infarct. (5) Calcification of neoplastic masses. (6) The tip of a vertebral transverse process may be so much more dense than the rest of it that it may suggest a calculus. Areas of calcification in the body of the twelfth rib may be misleading. (7) Areas of fibrosis in the spleen, which may overshadow the left kidney. (8) Calcification of a blood-clot; such are frequently found as phleboliths. (9) Calcification due to localized arteriosclerotic processes, so-called arterial plaques. (10) Calcification about any foreign body. (11) Shadows cast by fibromata, calcified fibroids, dermoid cysts, extensive scars, subcutaneous moles and warts, must be considered in the differentiation. It is unnecessary to state that artefacts which might occur in plate or film must be excluded.

All of our plain roentgengrams are made on the Bucky-Potter diaphragm, using duplitized films which include the entire body trunk and pelvis. Direct scrutiny of such a film shows at a glance the relationship of an area of increased density to any or all structures in or behind the peritoneal cavity. Films where the ureters and kidney pelves have been outlined with sodium bromid solution can then be directly compared to the plain plates. We have been using a 15 per cent. solution of sodium bromid for urographic purposes. Similar solutions of sodium iodid or thorium nitrate are also used with excellent results. The solution is always freshly prepared and sterile.

The steps necessary for the proper differentiation of a shadow in the kidney region are as follows: The plain plate has shown a possible calculus. There may or may not be symptoms referable to the urinary organs. Cystoscopic examination and catheterization of the ureters is done and radiographic catheters passed for a distance of 26 to 28 cm. from the ureteral orifices in the bladder.

Another flat plate is made. This will show the relationship of the shadow to the ureter and kidney pelvis region. In this regard it is sometimes helpful to make stereoscopic pictures at this time. Without allowing the patient to shift position, the sodium bromid solution is gently injected through the ureteral catheter. From 8 to 10 c.c. will ordinarily suffice to definitely outline the ureter and pelvis. Another film is made. The bromid solution is then returned into the syringe by suction. Another plain plate is taken at this time to demonstrate the possible coating over of the calculus with bromid solution, thus bringing out a contrast in density which might have been overshadowed by the picture of the completely filled pelvis.

I will not go into further detail with you today concerning the interpretation of pyelographic data. I might add, however, that this detail study is indicated even where we have sufficient reason to believe that the shadow is definitely in the urinary tract. Why? Such pictures will demonstrate first of all whether the calculus is in the pelvis, calix, or parenchyma. It will demonstrate evidences of kidney distention, and by study of possible filling defects, in conjunction with careful individual kidney function tests, will indicate whether attempts should be made to preserve the organ or whether its removal is rendered advisable. It will further assist the operator by showing him whether the renal pelvis is normal or "intrarenal." In the first instance a pyelotomy will be indicated, in the latter a partial nephrotomy will often be necessary.

It is beyond consideration that in a community with modern medical facilities any patient would be operated upon for a renal calculus without first catheterizing that kidney and determining its functional capacity. It is the only method which can give absolute indication as to whether that organ should or should not be removed. Much needless misery has been caused by leaving a hydronephrotic, pyonephrotic, or "stone-forming" kidney after the removal of a calculus in incompletely studied cases. On the other hand, many kidneys have been unnecessarily sacrificed because their gross appearance at operation gave the *impression* that they were functionally decadent.

I wish to briefly review 2 very instructive cases which are illustrative of several points just mentioned.

Case I.—An unmarried Jewish male, aged thirty-six years, presented himself October 25, 1921. He had just come from

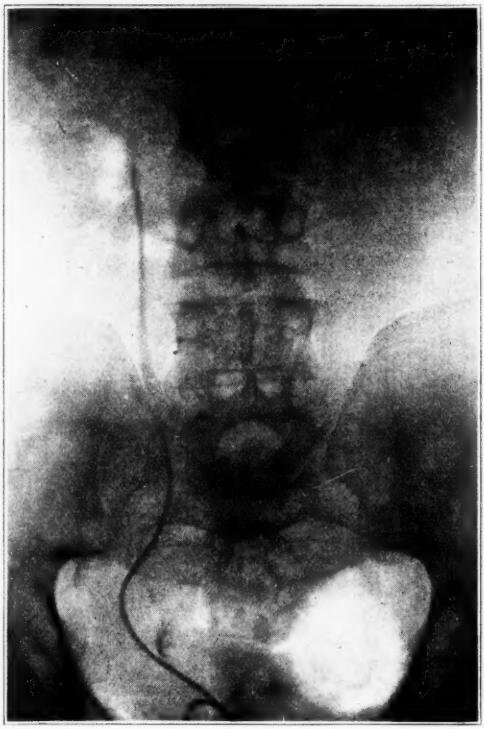

Fig. 76.

another hospital where three weeks before he had been subjected to a laparotomy for supposed gall-stones. No gall-stones had been found and the gall-bladder had apparently appeared normal, as it was not disturbed. The appendix was removed. On the third day postoperative the patient had a severe pain

in the right upper abdomen, which was typical of his former complaint.

His past history was unimportant. For three years he had been troubled with recurrent attacks of "knife-like" pain in the upper right abdomen. The pain was severe and required morphin for relief. Radiation of colic most frequently took place to the right lumbar region or to the right scapular region posteriorly. It had never radiated into the groin or thigh. There had never been any associated nausea or vomiting, but fever frequently accompanied the pain. No chills or gastro-intestinal irregularities. No frequency or other urinary symptoms. Repeated urinalyses had been negative for blood, pus, or albumin.

Flat roentgenograms showed two areas of increased density in the gall-bladder region (Fig. 76).

Physical examination revealed nothing abnormal except pain on deep bimanual palpation of the right upper abdomen. The right kidney could not be felt and there was no mass suggestive of an enlarged gall-bladder. Blood-count normal. Urine normal. Two-hour phthalein test yielded only 45 per cent. Blood urea nitrogen was 16 mgm. per 100 c.c.

Would not the surgeon in this instance have been more fair to this patient if he had definitely tried to identify the exact location of these shadows?

Cystoscopy showed a normal urethra and bladder in every respect. The left ureter was easily catheterized with a No. 6 French catheter for a distance of 28 cm. Clear, normal urine was obtained from this kidney. Dye appeared in three minutes after intravenous injection, and 18 per cent. was excreted in the first fifteen minutes.

It was impossible at the first cystoscopy to pass even the smallest bougie into the right ureteral opening. After three subsequent dilatations the right ureter was catheterized 10 cm. with a No 6 French catheter. A rapid flow of limpid urine resulted, and 40 c.c. was drained in three minutes. No dye appeared after intravenous injection. A right ureteropyelogram (Fig. 77) demonstrates the exact pathology: a long-standing ureteral stricture with subsequent gradual hydro-ureterone-

phrosis and calculus formation. Right nephrectomy completely relieved the condition and verified the diagnosis. A recent report from the patient is to the effect that he is absolutely well. He is doing hard manual labor in the oil fields.

Fig. 77.

Needless surgery and professional chagrin would have been avoided if the assistance of urologic methods had been obtained in the first instance.

Case II.—A Jewish widower aged fifty-two years presented himself March 24, 1923, complaining of frequent attacks of

severe knife-like pain in the right upper abdomen. The cause of this discomfort had repeatedly been diagnosed as "gall-stones" and "inflammation of the gall-bladder." The pain localized itself to the right upper abdomen along the margin of the ribs. It did not radiate, and after a very distressing attack the upper abdomen was very sore on deep palpation pressure.

The patient had been subjected to these attacks for over eight years, but more recently the severity of the pain and the increased frequency of "acute" attacks caused him to decide to "have something done about it." The day before he presented himself he had been examined and a "gall-bladder operation" advised.

Physical examination was essentially negative. The entire upper right quadrant was "sore" on deep palpation, but no masses could be felt in any part of the abdomen. The kidneys, liver, and spleen were not felt. There was no pain on pressure in the right costovertebral region. The genitalia were normal. Reflexes normal. Rectal examination showed no abnormality.

Repeated urinalyses showed no albumin, sugar, or microscopic abnormality. Blood Wassermann negative, cell counts and differential normal.

Flat roentgengrams of the abdomen showed the left kidney shadow in an apparently normal position, but slightly elongated. The right kidney shadow was very much enlarged and measured 11 cm. from hilus to periphery. In the area just below and median to the central portion of the right kidney outline there was an irregular shadow of uniformly increased density about 3 cm. in diameter, and 2 cm. below and to the right of this single shadow there were several irregular areas of increased density from 2 to 5 mm. in diameter. These smaller shadows were not nearly as dense as the single large one.

Despite the absence of clinical evidence pointing to disease of the urinary tract, it was deemed advisable to localize and identify these shadows which might presumably be in the gall-bladder. On cystoscopic examination the bladder and urethra were normal in every respect. No. 6 French ureteral catheters were passed 30 cm. on either side. There was a rapid flow

of limpid urine from the right catheter. By suction on the right catheter I was able to withdraw 40 c.c. of urine from the renal pelvis. This in itself demonstrated the presence of a hydronephrosis. On the left the urine was normal in flow,

Fig. 78

amount, and character. Intravenous phenolphthalein injection showed an appearance time of three minutes on the left and none appeared from the right for twenty minutes; 35 per cent. was excreted from the left kidney in the first twenty minutes.

Plain roentgenograms with the opaque catheters in place show the large heart-shaped shadow just above the tip of the right catheter. It appears to lie in the same plane, but this does not prove conclusively that it is in the kidney (Fig. 78);

Fig. 79.

15 per cent. sodium bromid was slowly injected through a syringe until 20 c.c. had entered. The patient complained of no pain. In the right pyeolgram it is then seen that the bromid solution passes around the large dense shadow, and then on to

outline a very irregular kidney pelvis, with entire loss of calyx outline. The left pyelogram was made with 8 c.c. of bromid solution. It was normal in every respect.

The diagnosis in this instance was clear.

There existed a right hydronephrosis with a large renal stone partially blocking the pelvis. It was assumed that the smaller shadows were probably stones or small centers of calcification within the lower portion of the kidney tissue. But there was no absolutely definite method to exclude them as possible gall-stones superimposed upon the kidney shadow.

On March 28, 1923 the right kidney was exposed through a lumbar incision and the diagnosis of multiple renal calculi in a very large hydronephrosis was verified. The kidney was easily removed, and on April 15, 1923 the patient left the hospital in excellent condition. On more closely examining the specimen to check back on the nature of the smaller shadows I found that some were due to definite calculi formation, while others were obviously cast by calcified plaques lining the lower portion of the hydronephrotic sac.

Please bear in mind that I am demonstrating these cases not as unusual instances at all. They are most common, but, unfortunately, routine urologic investigation is rarely deemed necessary, and many patients are subjected to laparotomy without an accurate diagnosis having been made. Remember that urinary symptoms or positive urine findings may be lacking, and yet definite pathology exist in these organs. This pathology is often the cause of pain and is frequently overlooked.

CLINIC OF DR. MILTON M. PORTIS

ST. LUKE'S HOSPITAL

THE cases I am presenting to you today are from my private service at St. Luke's Hospital.

PEPTIC ULCER

This woman is fifty-four years old. She came to the hospital complaining of acid eructations, vomiting, and pain in the upper abdomen. She also complains of weakness.

Present Illness.—Eight weeks before entering the hospital the patient, after eating a meal of clam chowder and oranges, felt uncomfortable, had acid eructations, became nauseated, and then vomited. Since that time her stomach has been giving her almost constant trouble. Belching has been frequent and it has been accompanied by a sour taste. She has vomited once or twice almost every day, but has had an occasional free period from vomiting lasting for a day or two. The vomiting is preceded by nausea for several hours. No special food seems to distress her. She does not get any relief on taking food. Vomiting may occur only once during the day, but on other days she has vomited one or two hours after every meal. At no time has there been any sign of blood in the vomitus. She has an almost constant gnawing sensation, which is accompanied at times by pain of a sharp and boring character. These severe attacks of pain are relieved by vomiting. Pain is felt in the upper abdomen and is frequently of greatest intensity on the right side near the costal margin. It is referred into the back and upward between the shoulder-blades. Alkalies give some relief, but it does not last long. She never awakens at night with the pain, but is nauseated on awakening, and at times

vomits food taken the night before. She is very nervous and complains of palpitation. She has lost 15 pounds in weight and has grown progressively weaker. This she attributes to the lack of nourishment. She has never noticed any tarry stools. The pain and abdominal discomfort are not relieved by bowel movement.

Past History.—She has had chronic articular rheumatism with exacerbations for many years. Six years ago she had an abdominopelvic tumor removed, of which she does not know the nature. She has not menstruated since that time. She has been constipated for many years and in recent years this has become very stubborn.

Family History.—Father died of pneumonia; mother died of old age. One brother died of kidney trouble. Her husband died of spinal trouble which was associated with some irritative motor lesion. She is not very definite concerning the nature of the trouble. She has never been pregnant.

Menstrual History.—Began at eleven years. Menses were regular without any pain until the menopause, which was induced by the operation six years ago.

Physical Examination.—The patient is highly nervous and moderately well nourished. Her hair is thin, dry, and gray. Pupils are normal. There are many teeth missing and most of the remaining ones contain fillings. There is no evidence of caries. Tonsils are small and ragged, but do not show any free pus. Submaxillary and cervical glands are moderately enlarged. They are discrete, firm, and not tender.

The lungs show emphysema. There are no râles. The apices are normal. The heart shows slight enlargement. The apex is in the midaxillary line. The second aortic tone is accentuated and there is a systolic murmur heard loudest over the base. The blood-pressure measures 130 systolic and 75 diastolic. The epigastrium is tender and the patient resists deep palpation. No mass can be felt. There is some tenderness in the right upper quadrant, but no evidence of rigidity. The gall-bladder cannot be palpated. The patellar reflexes are increased. The skin is dry and the subcutaneous fat is well retained.

The urine did not show any albumin, sugar, or casts. The reaction was acid.

Blood examination showed 3,620,000 red cells, 9000 white cells, hemoglobin of 75 per cent. The differential leukocyte count showed small mononuclears 20, large mononuclears 1, indentate nucleus 2, polymorphonuclears 71, eosinophils 2, and marrow-cells 4. The blood Wassermann test was clearly negative.

The stools show the presence of occult blood. The stomach contents show a free acid of 15 and total acid of 35. There was a strong reaction for occult blood. Sarcinæ were present in large amounts. The emptying time of the stomach is greatly delayed, food being present for twelve hours.

On entrance the temperature of the patient was 98.4° F., pulse 80, and respirations 20. She has not had any increase of temperature since entrance.

An *x*-ray examination made by Dr. Jenkinson shows a large dilated stomach with active peristaltic waves. There are two penetrating ulcers on the lesser curvature in the pars media. The pars pylorica is irregular on both curvatures and could not be filled out. The duodenal bulb could not be filled out. At the end of six hours the stomach contained two-thirds of the original contents.

Because of the high-grade motor insufficiency, the multiple ulcers, and the possibility of malignancy I considered the case to be a surgical one rather than one which should be given medical treatment, and referred the patient to Dr. L. L. McArthur for operation. He found two ulcers on the lesser curvature and a pyloric stenosis with scar formation, probably associated with a previous ulcer. In addition, the gall-bladder was found full of stones. There was no evidence of malignancy. The gall-bladder was removed and the pylorus was resected as well as the lesser curvature ulcers, and a gastrojejunostomy was done.

The patient made a good recovery.

Peptic ulcer is properly considered to be a single lesion. It is only rarely that more than one ulcer occurs at the same time. However, two or more ulcers may be present simultaneously, and judging from the literature this takes place more frequently

than is commonly supposed. The multiple ulcers may be in the stomach or the duodenum, or in both places. In the days before the use of the *x*-ray the diagnosis was made only in the operating-room or dead house. Now the Roentgen ray enables us to make such a diagnosis with accuracy before operation.

Rokitansky states that among his 79 ulcer cases, at autopsy 17 presented two or more open ulcers. Brenton, in an extensive study, concluded that two or more ulcers were present in 1 out of every 5 ulcer cases; in other words, about 20 per cent. of the cases were multiple. He reported 463 cases of ulcer, of which 97 had more than one. In the Mayo Clinic in a series of 638 cases of gastric ulcer, 28, or 4.5 per cent., had more than one present. Fenwick, in an exhaustive study of postmortem records, analyzed 876 cases as follows: One ulcer present in 698, or 80.5 per cent.; two ulcers present in 105, or 12.1 per cent.; three ulcers present in 27, or 3.1 per cent.; four or more present in 37, or 4.2 per cent. These were all cases with active or open ulcers. If the scars of healed ulcers were also counted then the percentage would run much higher, for ulcers heal and recur at the same or other spots in the same region. It is not uncommon to meet in the same stomach open ulcers in the scars of healed ulcers. Numerous ulcers are usually of the acute type. When two or three are present they are most frequently of the chronic variety. Most of the cases diagnosed by the *x*-ray before operation are of the chronic type. The number simultaneously present rarely exceeds 5. Bolton states that acute ulcers occur chiefly in three types of cases: Acute infective processes, chronic infective processes, and where there is no obvious infection. In the first group all the ulcers are in an open condition and scars are rarely seen, while in the second group it is not uncommon to find partially healed ulcers and scars together with recent ulcers. The acute gastric ulcers tend to recur and commonly heal. In rare instances the number of ulcers present in the stomach is excessive. Hewitt reports a case with sixty ulcers in the stomach. Berthold mentions one with thirty-four deep perforations in the stomach. Osler reported one with thirty-four ulcers in the stomach, and Lange described one in

which the ulcers were too numerous to count. The mucosa in such cases is riddled with small ulcerations, and usually such cases are associated with septic infections and secondary syphilis.

Ulcer in the duodenum is usually solitary, but it, like gastric ulcer, may be multiple. Merot found a single duodenal ulcer in 82 per cent.; two in 13.5 per cent., and three or four in 4.5 per cent. Carmen states that 11.5 per cent. of gastric ulcers have associated duodenal ulcer.

The histopathology of the multiple peptic ulcer resembles that of the usual findings of a single duodenal or gastric ulcer. In chronic duodenal ulcer there is usually a superficial destruction of the mucosa with moderate involvement of Brunner's glands. However, there is practically no destruction of the underlying muscularis and serosa. There may be a lymphocytic infiltration of the adjacent tissue and a moderate endarteritis. Changes in the ganglion cells have been noted by several writers. The chronic gastric ulcers usually present a quite different, although characteristic, picture. The mucosa ends abruptly in the region of the ulcer and the borders are sharp and project to the floor of the ulcer. There is usually an entire absence of mucosa, submucosa, and muscularis at the site of the ulcer. The floor of the ulcer is formed by granulation tissue and thickened serosa. There may be a definite subacute inflammatory reaction in the adjacent tissues with an associated arteritis and involvement of the ganglion cells. In acute ulcers the involvement is usually limited to the mucosa. It is in this type that the acute perforation is more apt to occur. Moynihan has called attention to the fact that perforating ulcers are at times multiple, and he advises that as soon as the ulcer first discovered is sutured, that a rapid survey of the whole stomach is desirable in order that any other ulcers may be laid bare. In the majority of cases the second ulcer was found on the posterior surface at a point exactly opposite the first. It is important in all *x*-ray studies for ulcer not to be satisfied with finding of one, but to look carefully for another in the stomach and duodenum. Small ulcers are very difficult to detect and are often missed unless an especially careful search is made.

GANGRENE OF THE TOES THE RESULT OF ENDARTERITIS OBLITERANS

THIS man is sixty-two years of age. He entered the hospital complaining of pain in the right foot and toes and difficulty in walking. About four months before entrance the third and fourth toes of his right foot began to pain him at the very tips, just under the nails. The pain was of a burning and shooting character. Shortly after the onset he noticed that the toes were bluish and finally became black to the second joints. With the change in color the pain became worse, and he has been unable to sleep because of it. The pain is accompanied by an intense sensation of burning. The patient has for a number of years been accustomed to arise at night to urinate, but the total amount during the day is not excessive. He does not complain of thirst. He has no cough. A week after the onset small blisters formed and there was slight swelling of the toes of the right foot. The fourth toe opened spontaneously under the nail and there was a discharge of a serous fluid, and within a day the third toe also opened up and discharged a similar fluid. The fourth toe gradually healed up, but the third toe remained open and the nail dropped off and pus began to discharge. The pain is now continuous and is especially bad at night. He has no appetite and feels weak and is quite nervous. His bowels are constipated and have been so for many years.

Past History.—Usual diseases of childhood; appendectomy twenty years ago; ulcer on right leg above ankle twenty-five years ago. He denies all venereal infections.

Family History.—Both parents are dead; he does not know the cause. He has 7 children living and well. His wife is in good health. There is no history of diabetes in the family.

Habits.—He uses coffee and tea freely and tobacco excessively. Alcohol is indulged in moderately.

Physical Examination.—The patient is poorly nourished and looks older than he says. He has an expression of pain.

His pupils react promptly to light and accommodation. Reflexes are all normal. The thyroid gland is not enlarged. The teeth are in poor condition. The gums are soft and spongy and the tongue is coated. The tonsils are not diseased.

There is a slight dulness over the right upper lobe. Breathing is roughened in this area. A few moist râles are heard. The rest of the lung findings are negative. The heart is moderately enlarged. The pulse is regular. The second aortic tone is accentuated. There is a loud systolic murmur heard over the base and transmitted into the vessels of the neck. Blood-pressure measures 150 systolic and 74 diastolic.

He has an appendectomy scar, but otherwise the abdominal findings are negative. There is an area on the plantar surface of the third and fourth toes of the right foot which is black and dry and measures 1 cm. in width. There is some edema in the surrounding area and cyanosis. The left foot is pale and both feet are cold. When the right foot is kept in a dependent position a distinct flushing is seen of the entire skin. The same thing occurs when the left foot is put in a dependent position. Pulsation cannot be felt in the dorsalis pedis or in the posterior tibial of the right foot, and it is barely perceptible in these vessels in the left foot.

The urine examination shows a faint trace of albumin and a few hyaline casts. There was no sugar present. The phenolsulphonephthalein test was 150 c.c. at the end of two hours and showed a total of 28 per cent.

Blood examination showed 4,100,000 reds, 10,500 white cells, and hemoglobin 86 per cent. The differential count shows 22 per cent. small mononuclears, 70 per cent. polymorphonuclears, 4 per cent. transitionals, and 1 per cent. eosinophils. Blood-sugar was 0.1003 per cent., blood urea nitrogen 18.54 mg. per 100 c.c.; non-protein nitrogen was 37.46 mg. per 100 c.c. The blood Wassermann was clearly negative. The stool did not show any occult blood.

While in the hospital the patient ran a normal temperature. He was seen in consultation with Dr. L. L. McArthur, who advised against any surgical intervention and thought that

medical treatment should be carried out. The foot has been kept elevated under aseptic conditions and the patient is on a low protein diet. The use of tobacco is stopped and he has been taking modified Ringer's solution. This is made by dissolving 10 grains of potassium chlorid, 10 grains of calcium chlorid, 6 drams of sodium chlorid, 10 grains of sodium bicarbonate, and 50 grains of dextrin in a gallon of distilled water. The patient is taking 2 to 4 quarts of this special water daily. We are urging him to take as near 4 quarts as possible, but some days he is unable to take the full amount. The drinking of this liquid is forced between meals, during the day and during the night, so as not to interfere with his digestion. This is a hypotonic solution like Ringer's solution, but it is rendered more palatable by the addition of the sugar. The reason for using it will be discussed later in the clinic.

Already a marked improvement has taken place in the patient, and I think the outlook is a good one for staying the progress of the disease and repairing the damaged tissues, so that an amputation will not be necessary. It will be very slow in getting granulation tissue over the necrotic areas, but this has started very well already and the progress is a good one for the complete repair. He will be kept upon this liquid for many months and the use of tobacco will be forbidden. Gradually he will be allowed some proteins in his diet and his activity increased as his condition permits.

THROMBOSIS OF THE VESSELS OF THE LEG AND OF THE MESENTERIC VESSELS

THIS woman is thirty-seven years of age. Three weeks before her entrance into the hospital she felt a numbness in her right foot and it was weak. An irregular pain appeared, especially in the toes. She noticed that her skin was blanched and that the foot was slightly swollen on the instep. Within a few days the toes began to swell. At times the color was bluish and at other times perfectly white. With the swelling the pain increased and became constant, especially in the toes, and then in the instep. This pain has grown so severe that it has interfered with her sleep and it has been necessary to take opiates for relief. The pain at times extends into the calf of the left leg, but it is not constant and has not been present for several days. With the onset of the severe pain the toes of the right foot became bluish red, and this cyanosis extended on to the sole of the foot. She has been very nervous, fretful, and fearful. She complains of palpitation and insomnia. Her appetite is poor. She has had no cough or night-sweats, nor has she had any headaches. She has vomited at times, but does not have any distress after eating.

Past History.—She had the usual diseases of childhood. She has had an enlarged thyroid for many years, associated with nervous symptoms. She consulted the Mayo Clinic three years ago because of it, and was told that an operation on the thyroid was not necessary. Her nervous symptoms have continued and from time to time she has had tachycardia. However, she felt fairly well until three months ago, when her mother died. Since that time her nervous symptoms, palpitation, and melancholia have persisted. When she was at the Mayo Clinic a small lump was removed from underneath the left breast, which they told her was not malignant. For the past six years she has had headaches of a migraine nature, but these stopped suddenly six months ago and have not given her any trouble since.

Family History.—Negative for diabetes, tuberculosis, and malignancy.

Marital History.—She has been married fifteen years. Her husband is living and well. She had one miscarriage two years ago which was spontaneous, and there have been no other pregnancies.

Menstrual history is normal.

Habits.—She uses coffee and tea freely and leads a sedentary life.

Physical Examination.—She is fairly well nourished, but looks distressed and ill. Her hair is light brown in color, with beginning grayness. The pupils react normally to light and accommodation. She does not show any of the characteristic eye muscle tests for Basedow's disease. Most of her teeth are missing and the few remaining ones are not in good condition. The tongue is coated and the breath is foul. The tonsils are very small and ragged and the pillars show some injection. The posterior cervical glands are small and firm. The thyroid gland is enlarged in all of its lobes. It is firm and not tender.

The chest is poorly developed. Expansion is equal on both sides. The lungs do not show any areas of dulness and no râles are heard. The heart shows a slight enlargement in the transverse diameter. The pulse is rapid, but regular, and there are no murmurs present.

The abdomen is flaccid, the liver is not enlarged and is not tender. The spleen cannot be palpated. There is no evidence of hernia. Pelvic examination did not show any abnormality.

Both feet are cold to the touch and the left one is pale. The right one shows marked hyperemia and cyanosis, especially involving the toes. There is swelling over the base of the first metatarsal and over the second and third toes. This region is extremely tender. On the tip of the third toe the skin shows some beginning necrosis. It is dry. The reflexes are sluggish, but present. The pulse cannot be felt in the dorsalis pedis, nor can it be felt in the posterior tibial on the right side. The pulse is very weak in these vessels on the left side.

x-Ray examination made by Dr. Jenkinson shows some scle-

rosis in the vessels of the right foot, but not of the left. There is some evidence of substernal thyroid. The lungs show an increase of peribronchial markings, but do not show any other changes. The left heart measures 8.2 cm., right heart 4.3 cm., great vessels 7.3 cm. The transverse diameter of the chest is 27 cm.

The temperature on entrance was normal, whereas the pulse ranged from 100 to 120, with respirations from 22 to 26. The urine shows a trace of albumin with a few hyaline casts and a few red blood-cells. There is no sugar present. The stool did not show any abnormal findings.

Phenolsulphonephthalein test showed a return of 50 per cent. in two hours. The blood urea nitrogen was 21.2 mg. per 100 cm., non-protein nitrogen was 51.64 mg. per 100 cm., blood-sugar was 0.102 per cent.

The blood examination shows 4,100,000 red blood-cells; 9800 white cells, and hemoglobin 85 per cent. The differential leukocyte count shows 17 small lymphocytes, 6 large mononuclears, 5 indentate nucleus, 72 polymorphonuclears. Blood Wassermann was clearly negative.

The third day after her entrance to the hospital the patient was feeling more comfortable and was having less pain, when suddenly she complained of an agonizing pain in the epigastrium. This was associated with nausea and she vomited several times. The pain was of a griping nature and very soon was followed by several watery movements. There was generalized tenderness in the abdomen, especially marked in the epigastrium. There was no evidence of rigidity. The tender area in the epigastrium corresponds with the region in which she feels the severest pain. Nausea has persisted, but she has not vomited since the onset. The vomitus was bile stained and did not have a fecal odor. The bowels have not moved since the onset of this severe pain. The leukocyte count before the onset was 9800; four hours later it was 23,600 and two hours later 39,000.

Another count made one hour later was 42,600. The patient had an anxious look and was in great distress, suffering severely with pain. Dr. H. M. Richter saw her in consultation

to decide the question of an operation. In the differential diagnosis mesenteric thrombosis, perforation, acute pancreatitis, and disease of the gall-bladder or appendix came up for consideration. The blood-pressure rose to 210 systolic and 110 diastolic. This increase of blood-pressure helped to rule out the question of pancreatitis. There was no localized tenderness over the region of the appendix or gall-bladder, and hence it seemed unlikely that these could be the cause of the acute picture. The absence of rigidity, of free fluid, and the loss of liver dulness all spoke against perforation. Mesenteric thrombosis was considered to be the probable cause of the acute sudden picture. In the hope that an intestinal resection might be done and also considering the possibility of atypical perforation, a laparotomy was performed.

On opening the peritoneum there was no evidence of free fluid and likewise no fat necrosis was seen. The entire small bowel was dark in color and in places almost black. There was no evidence of pulsation in any of the mesenteric vessels. Some of these vessels felt firm and cord-like. Because of the wide extent of the thrombosed area resection was impossible. The abdomen was closed. The patient, as you observe, is still in shock. The pulse is rapid and of poor quality. She has frequent emesis, and in spite of all measures that we can carry out the outlook is hopeless and the end is only a matter of hours.

The 2 cases here presented show trophic disturbances of the foot which in one case advanced to gangrene of the toes. Undoubtedly there is some interference with the circulation to cause these findings.

The pathology of this condition is variable and there are a number of things that can cause it. There has not been a history of trauma in either case. The blood Wassermann in both cases is negative. Neither of these patients shows any clinical signs of syphilis. The old man shows definite evidence of generalized arteriosclerosis, but this is not present in the case of the woman. Both started with pain in the foot and legs, both complained of numbness and coldness, and in both cases the pain became worse and was unbearable even when the legs were

kept at rest. Blanching of the affected foot changed later to cyanosis. The clinical story and picture in these cases rules out Raynaud's disease. There is no evidence in the urinalysis or in the blood-sugar of the presence of diabetes. Buerger's disease, or thrombo-angiitis obliterans, is commonest in young Jewish males, especially in those who use cigarettes freely. It affects usually the lower extremities, but at times also the upper. Buerger considers this to be of infectious origin, producing an inflammation of the walls of the arteries, a condition which goes on to thrombosis and obliteration of the lumen. In the case of the man the trouble is most likely an endarteritis obliterans of the vessels of the foot associated with his generalized arteriosclerotic changes. He was relieved by giving fluid which reduced the viscosity of the blood, and in this way helped his circulation. In the case of the woman thrombosis has undoubtedly occurred in the vessels of the right leg and also in the mesenteric vessels. The origin of this in her case is obscure. A careful postmortem will be made and will undoubtedly determine the exact nature. Neither of these cases belong definitely in the group described by Buerger, although it is possible that some infection may be responsible for the trouble and that the final analysis may place them in the Buerger group.

CLINIC OF DR. WALTER W. HAMBURGER

MICHAEL REESE HOSPITAL

ACUTE CARDIAC PSYCHOSES: ANALYSIS OF THE TOXIC AND CIRCULATORY FACTORS IN 5 CASES OF ACUTE CONFUSION

DURING the past year I have been particularly impressed with the development, in cases of advanced and recurring heart failure, of severe mental symptoms, so severe in several cases as to justify calling them acute cardiac psychoses. In certain of these cases these mental symptoms have largely dominated the clinical picture, and because of this I thought you might be interested this morning in going over them briefly with me in an attempt to analyze the significance of such acute psychoses in heart disease from the viewpoint of diagnosis and treatment.

The literature of the subject is extremely meager, and it is for this reason also that one may be justified in presenting them. The crucial point of discussion in the literature on these acute mental confusions revolves about the question as to the underlying etiology. For example, David Riesman,[1] in an interesting discussion before the Association of American Physicians in 1920, sums up the causes of the cardiogenic psychoses, as he calls them, under six divisions: First, an accidental one occurring in an individual of psychogenic make-up; second, concomitant disease of the kidney, uremia; third, acidosis; fourth, various drugs and poisons, notably alcohol, digitalis, etc.; fifth, liver and intestine, "swamping of the system with toxic material"; sixth, direct disturbance of the cerebral circulation affecting the higher centers.

[1] Riesman, David: Acute Psychoses Arising During the Course of Heart Disease, Amer. Jour. Med. Sci., 1921, clxi, p. 157.

Romberg[1] notes acute psychoses in heart disease with severe disturbance of the cardiac mechanism. They are chiefly of a depressive character and are associated with abrupt changes in the patient's demeanor—a desire to change his position in the ward or to go into a new ward because of a fear of his ward companions, a sudden desire to change physician or nurse. He calls attention to the fact that this change in mentality may persist a long time—in one case of mitral stenosis over one year. He feels, however, that it seldom occurs, noting it only 13 times in 1200 cases in the Leipsig Clinic. It is always of serious prognostic significance, occurring usually in patients over thirty-five years of age, and usually at the time of a decrease in cardiac efficiency, otherwise independent of the type of heart involvement. Many of these patients feel they have been poisoned and are being persecuted; they stop eating, they refuse their medicine. Later they have visual and auditory hallucinations. They see people sitting on their bed, or peeking at them through the door and windows, mobs pursue and threaten them. They become severely excited and endeavor to get out of bed. They attempt to throw themselves from the window or attempt suicide in other ways. They suffer severely with insomnia. In the early stages they have an insight into their hallucinations, thinking possibly they have dreamed them. Later they lose this insight and are totally disoriented. Curiously enough the delirium often clears just before death. Romberg differentiates these cardiac psychoses from the alcoholic psychoses and from uremia. Head[2] believes these hallucinations are especially frequent in aortic disease.

Case I.—The first patient I wish to present to you is a merchant, forty-seven years of age, a resident of Mississippi. He was quite well up to April, 1921, when he developed a sudden tachycardia of unknown origin. Somewhat later it was thought he was suffering with an early Basedow's disease, and on ex-

[1] Romberg: Krankheiten Des Herzens, 3d ed., p. 148.

[2] Head, H.: Certain Mental Changes that Accompany Visceral Disease, Brain, London, 1901, xxiv, 345.

amination there was found a metabolic rate of 53 per cent. plus. In July of the same year he consulted the younger Kocher, in Berne, who agreed with the diagnosis of hyperthyroidism and ligated the superior thyroid artery on the right side. Following this he improved markedly, remaining fairly well until December, 1921, when he suddenly developed a rapid type of auricular fibrillation, which has persisted ever since.

I first saw him in September, 1922, at which time my diagnosis was hyperthyroidism with thyrotoxic heart and chronic auricular fibrillation and myocarditis, with early heart failure. After a short period of rest in bed and moderate amounts of digitalis the apical pulse-rate came down to an average of 80 to 84 per minute. He was then given small amounts of quinidin to control the fibrillation of his heart, without effect. The quinidin was promptly discontinued because of evident increase in restlessness, nervousness, and what I thought was a slight increase in his decompensation symptoms. After another few days he returned to his home in Mississippi. Two weeks later he re-entered the hospital because of a severe recurrence of his heart failure and was then treated for a long period with complete rest and digitalis, making a very satisfactory improvement. November 9th digitalis was stopped and quinidin was again started, and from the 9th to the 15th of November a total of 7.4 grams of quinidin was given. The adjoining table (Table 1) contains an outline of data regarding his medication, pulse-rate, important mental symptoms, etc. As you will see in this chart, symptoms of profound mental confusion occurred forty-eight hours after the discontinuance of the quinidin and persisted in an extremely severe form for two weeks. Because of his severe mental disturbances I asked Dr. Sidney Kuh to see him from a neurologic standpoint, and he made the following notes on the 22d:

"Patient has a relatively clear memory for things which happened a few years ago, but very poor for recent events. There is no disturbance of the tongue. He understands perfectly. There is no loss of muscle power. Abdominal reflexes not obtained. There is marked instability of vasomotor system.

TABLE 1

Case I. Mr. R.

Medication

9/22/22: Quinidin, 0.2 gm. b. i. d.
9/23/22: Quinidin, 0.2 gm. five times daily—stopped. Followed by nausea, anorexia, pain in right eye, attack of dyspnea, irregular tachycardia.
10/17/21: Tr. digitalis, dr. j, every six hours.
10/21–24: Tr. digitalis, ♏xx, every six hours.
10/20–30: Tr. digitalis, ♏xv, t. i. d. p. c.
10/30 to 11/17: Tr. digitalis, ♏x, t. i. d. p. c.
11/9: Quinidin, 0.2 gm. b. i. d. = 0.4 gm.; pulse 80–84.
11/10: Quinidin, 0.2 × 7 = 1.4 gm.; pulse 80–98; pain in right eye, headache, nausea, tired; anorexia.
11/11: Quinidin? = 1.2 gm.?
11/12: Quinidin, 0.4 gm. × 3 = 1.2 gm.; pulse 88–100.
11/13: Quinidin, 0.4 gm. × 4 = 1.6 gm.; pulse 92–96.
11/14: Quinidin, 0.4 gm. × 2 = 0.8 gm.; pulse 98–136.
11/15: Quinidin, 0.4 gm. × 2 = 0.8 gm.; pulse 110–154.

7.4 gm. (irrational)

11/16: Tr. digitalis, ♏xxx.
11/17: Irrational; dazed, after three weeks digitalis and three days quinidin.
11/18: Confused and disoriented.
11/19: Out of bed; ready to go away; does not know where he is.
11/20: Out of bed; "They are coming after me."
11/21: Thinks he is on the train.
11/22: Thinks he is in New York; Cleveland; calls to boy he sees.
11/23: Thinks he has had an operation, result of accident.
11/24: Frightened; "must take precautions."
11/30: Realizes where he is for the first time in two weeks.

Knee-jerks equal, brisk, normal. There is a well-marked antero-retrograde amnesia.

"Diagnosis: Korsakoff syndrome. Of the known etiologic factors, only one is present in this case, *i. e.*, high blood-pressure. If quinidin can produce it, the fact has, so far as I know, not been recorded. The prognosis is entirely dependent on that of his physical condition. I doubt very much if there is any focal lesion in the brain."

Two of the most persistent hallucinations of this patient were that nightly his room was besieged by a mob of men who gathered outside the hospital and attempted to invade his room

and carry him off, and the angry excited voices which he could hear every night which kept him from sleeping. This hallucination persisted for weeks after he had in every other respect returned to normal. Likewise he was convinced that nightly his room was entered by both men and women, who disturbed his rest by talking, laughing, etc., during the midnight hours. For several days he was convinced that he had died and was in his grave and the events of wnich he spoke were occurrences of his after-life. This patient is still seriously ill (March, 1923), although now the mental symptoms have disappeared, with the exception of a certain instability of temperament with frequent unexplained variations in mood, demeanor, etc. Since the period of his acute confusion he has had three other attacks of severe decompensation, none of which have been accompanied by mental symptoms. It is difficult, therefore, to reach definite conclusions regarding the etiology of his confusional state, although it is rather strongly suggested that quinidin played an etiologic rôle, for in each of the other recurrences of decompensation he has had digitalis in about the same quantities, and in this one instance only had quinidin been given.

Quinin and its derivatives are not mentioned in the literature on toxic involvement of the central nervous system, where attention is called to the occasional acute poisonings and intoxication psychoses developing from alcohol, ether, chloroform, veronal, chloral hydrate, bromids, cannabis, nicotin, opium, morphin, cocain, lead, arsenic, mercury, sulphur, manganese, carbon dioxid, ergot, pellagra (maidismus). However, Cloetta[1] describes the so-called chinin rausch (quinin drunk), consisting of vertigo, clouding of consciousness, drunken-like excitement, later depression.[2]

Case II.—The second patient, a woman forty-nine years of age, entered the hospital July 31, 1922 complaining of dyspnea

[1] Cloetta: Die Vergiftungen durch alkaloide und andre Pflanzenstoffe, Handbuch der Inneren Medizin, vi, p. 706.

[2] Dr. L. J. Pollock (personal communication) states acute hallucination insanity has been described from quinin poisoning.

of six weeks' duration, precordial pain occurring at irregular intervals, anorexia, and exhaustion. There was nothing further notable in the history except the fact that she had had two miscarriages after the birth of her 3 children, of three and four months' duration respectively; that she drank 10 cups of either tea or coffee daily, and was accustomed to having a whisky toddy at night. My impression of her was that she was suffering from arteriosclerotic heart disease, chronic myocarditis, and advanced heart failure.

She was put on the usual routine of rest in bed, digitalis, etc. August 9th at midnight the intern notes, "She seems to be in a typical alcoholic (?) delirium; sees all sorts of things, makes queer noises, picks at the bedclothes, is quite irrational, acts very much like a D. T." Dr. Lewis J. Pollock, who saw her with me, made the following notes:

"Consciousness clouded moderately at times, seemingly disorientated as to place. Because of defect of consciousness, comprehension somewhat diminished; impressionability very poor and, as a result, memory for recent events very poor. Some falsification of sense perception, no insight as to existence of illness. Marked paucity of ideas and some lack of interest. Psychomotor activity somewhat increased. In general, the picture is that of a subacute confusion probably on a basis both toxic and circulatory."

August 21st death occurred very suddenly. In retrospect this patient probably also is to be classed as an acute cardiac psychosis, possibly on the basis of a chronic alcoholism.

Case III.—The third patient, a woman of sixty-four, had complained of dyspnea on exertion for years, swelling of the hands and feet, orthopnea, trembling of hands and arms, difficulty in walking, and weakness for five months. The dyspnea, she stated, had gradually increased during the last year. She was able to walk up one flight of stairs until about eight months ago, but during the past five months she has been so weak, exhausted, and "trembly" when trying to walk that she no longer attempts it, and had been practically constantly in bed for this

period. She has suffered from recurrent quinsy sore throat until tonsillectomy four years ago, and had influenza-pneumonia during the 1918 epidemic.

Her father had died of "hardening of the liver" at fifty-two and her mother from heart trouble at sixty-two; her husband of diabetes at sixty-four.

After observing her for a time my conclusion was that she suffered from generalized arteriosclerosis, chronic arteriosclerotic myocarditis with beginning heart failure, and paralysis agitans. The accompanying table (Table 2) gives the amounts

TABLE 2

Case III. Mrs. M. G., age sixty-four.

Medication

Tr. digitalis	℥xv	t. i. d.— 12/24/22 to 12/26/22	= m. 90
Tr. digitalis (Upsher-Smith)	℥xxv	q. i. d.— 12/27/22 to 12/28/22	= m. 200
Tr. digitalis (Upsher-Smith)	℥xv	q. i. d.— 12/28/22 to 12/30/22	= m. 120
		Six days	= m. 410

Effects

12/28: Nausea and vomiting, drowsy; pulse 60–72.
12/29: Severe headache, nausea and vomiting; pulse 72–92.
12/30: Severe headache, nausea and vomiting; pulse 90–96.
12/31: Severe headache, nausea and vomiting; pulse 94–108.
1/1/23: Anorexia; pulse 100–116.
1/2: Seems dazed, talks queerly at times; hallucinations during night; later irrational; sitting on floor; restless. Temperature 100° (R.); 4 stools; pulse 96–124.
1/3: Temperature 100.2° F.; pulse 108–124; respirations 26–32. Hallucinations, irrational, tries to get out of bed.
1/4: Temperature 100.4° F.; pulse 104–128; respirations 30–36. Irrational, muttering incoherently, imagines there are people in room, sits up in bed moaning.
1/5: Three attempts to get out of bed; four stools.
1/6: Three stools; vomiting, nausea.
1/8: Thinks she is on train; in Arizona; in undertaker's chapel, etc. Four stools; nausea and vomiting.
1/9: Depressed.
1/10: Crying and excited; believes she is in asylum; on ship; talks continually.

Impression.—Development of acute confusion synchronous with nausea, vomiting, diarrhea, increase in pulse, temperature and respiration, scant output of urine, cough, etc. An acute intoxication psychosis, either uremic, from digitalis, or both.

of digitalis and the days, showing that within six days she had received a total of 410 minims of Upsher-Smith tincture. Within three days after its discontinuance she seemed dazed and talked queerly at times, had hallucinations both visual and auditory, constantly attempting to get out of bed, at times extremely depressed, at others excited and irrational. Dr. Sidney Kuh, whom I asked to see her, concluded as follows:

"Memory for recent events poor. Mistakes the examiner for a man whom seemingly she has never met. Explains her getting up at night by saying she sees a child who is being attacked by a larger individual and feels it necessary to come to the aid of the attacked child. Does not quite distinguish dreams from reality, suggesting a consciousness and mentality which are more or less characteristic of senile dementia."

In this patient the development of the psychosis occurred coincidently with nausea, vomiting, diarrhea, increase in pulse, temperature and respiration, and scanty output of urine, and cough. This occurrence gives one the impression that this acute confusional state may have occurred on a toxic basis, initiated possibly by large amounts of digitalis.[1] This case is similar in many ways to the 2 cases reported by Carr,[2] who felt that the delirium was due in all likelihood to the toxic effects of digitalis given in large quantities. Duroziez[3] and H. O. Hall[4] believe that similar delusions may be caused solely by digitalis and may disappear when the drug is discontinued. It should be stated that this patient has made a complete recovery as far as her mental symptoms are concerned, and now has complete insight into and discusses her delusions with considerable acumen and humor.

[1] In a recent case of severe tic douloureux in a woman of sixty-five with generalized arteriosclerosis, hyperarterial tension and moderate cardiac decompensation, the use of only small amounts of digitalis was promptly followed by a mild acute confusion which persisted ten days.

[2] Carr, J. G.: The Toxic Effects of Digitalis, Annals of Medicine, vol. i, p. 548, February, 1921.

[3] Duroziez, P.: Du déliere et du coma digitaliques, Gaz. hebd. de méd., Paris, 1874, ix, 780.

[4] Hall, H. O.: The Hallucination of Digitalis: Does Digitalis Cause Hallucinations, Delirium or Insanity Under Certain Conditions? American Medicine, Philadelphia, 1901, 1, 1908.

Case IV.—Mr. J. W. H., sixty-six years of age, suffering with generalized arteriosclerosis and arteriosclerotic myocarditis, has had repeated recurrences of heart failure, the most recent one due to a right-sided coronary thrombosis, with severe angina and left ventricular infarction. In only one of these periods of decompensation did he have evidence of mental confusion. He uses neither alcohol nor tobacco. For thirty years he has been a sign-painter, although at no time showing evidence of plumbism. During his illness he suffered with severe Cheyne-Stokes breathing, which has persisted up to the present time. His mental symptoms consisted of restlessness, disorientation, excited and irrational talking, attempts to get out of bed, confusion as to time and place, sudden periods of lethargy without apparent cause, etc. It is extremely difficult to offer an adequate explanation for this one period of mental confusion occurring with an attack of severe decompensation, while other attacks of cardiac failure of equal or greater severity, in treatment essentially the same, developed and receded without evidence of mental confusion.

Case V.—The last patient I wish to speak of was seen several years ago. A man, forty-nine years of age, suffering with chronic interestitial nephritis with hyperarterial tension, chronic myocarditis, with advanced heart failure, and tabes dorsalis. This patient suffered with repeated severe abdominal and lower chest pains which for a time I thought might be the abdominal type of angina, but later considered them to be tabetic pains. In the attempt to control these abdominal cramping pains with various measures, atropin was given in physiologic doses. For instance, within thirty-six hours he received a total of 1/10 grain of atropin, given in 1/100 grain amounts every four hours from September 2 to 4, 1920. At this time the history notes: "The patient is irrational; has hallucinations; talks to imaginary people in the room; sees people on his bed; is restless and at times refuses to stay in bed; he talks irrationally during the greater part of the night, has constant hallucinations; is rational for a few minutes at a time. complains of feeling dizzy, and

believes he is on board a ship on the Hudson River." Dr. Peter Bassoe who saw him because of his mental confusion diagnosed a toxic psychosis, probably on the basis of belladonna-poisoning. This patient made within another three days complete recovery from his mental symptoms, only to die from advanced heart disease four months later.

It is interesting to note that of these 5 patients, 2 have died. The other 3 continue seriously ill, with, I believe, an extremely bad prognosis from the standpoint of partial or complete restoration of their cardiac efficiency. One feels then that the occurrence of these severe cardiac psychoses is, from a prognostic viewpoint, of grave significance. Romberg holds to the same view.

Summarizing the salient points of these 5 cases there are certain findings common to all. First, they all suffered from advanced and recurring heart failure and the development of the acute confusional state coincided with the period of severe decompensation. One must conclude, therefore, that the exciting cause of these psychoses, or at least a constant factor in them, is the presence of advanced heart failure.

Second, they were all patients over forty-five years of age and showed, for the most part, evidence of advanced arterial changes. It is likely that, in addition to the generalized arteriosclerosis which they presented, there was an associated cerebral arteriosclerosis with accompanying nutritional brain changes. Autopsies were not obtainable on the 2 patients of this series who died. However, the brain of a woman of fifty-five dying from generalized arteriosclerosis and advanced heart failure recently, who developed a transitory acute confusion shortly before death, showed large yellowish-gray atheromatous plaques along the course of the basilar vessels and extensive generalized edema of the brain with pressure flattening of the convolutions.

Third, all blood-pressure readings with one exception were over 170 mm. Hg.

Fourth, in addition to these findings, each case presented an additional etiologic factor, namely, digitalis, belladonna, quinidin, alcohol, and lead respectively. The dosage of the drugs used in

individuals with normal circulation and normal blood-vessels would not in all likelihood have been followed with untoward symptoms. One concludes, therefore, that the development of these confusional states may be due to the effects of these drugs on a circulation severely embarrassed, and on tissues with profound and wide-spread changes.

From the standpoint of treatment there is little to be said except possibly from a prophylactic standpoint. That is to say, that in patients over forty-five years of age, with severely embarrassed circulation, with arterial changes, localized particularly in the brain vessels, medication should be given cautiously and in amounts somewhat lower than the average doses. In the event of an acute confusion developing the prompt withdrawal of all presumably harmful medicines should be ordered. Restlessness and excitement should not be controlled with opium, hyoscin, or other sedatives if they can possibly be avoided, hydrotherapy being preferable. Romberg mentions the subcutaneous use of sodium luminal in doses of 0.3 to 0.5 and of scopolamin hydrobromid in doses of 0.0001 to 0.0003. He believes also that morphin increases the symptoms rather than controls them. Of importance are complete isolation; careful nursing supervision, with care that the patient is never left alone for a moment; institutional treatment; attention to nutrition, with frequent small meals with frequent variation; tube feeding in difficult cases; warm or cold packs; restraining sheet, etc. Paraldehyd in 1- or 2-dram doses two to four times daily is preferable to the other sedatives. Sweats and hot packs, the diuretic salts, low protein and salt-poor diet are indicated in patients with uremia.

CLINIC OF DR. CLIFFORD G. GRULEE

PRESBYTERIAN HOSPITAL

THE CARE AND FEEDING OF PREMATURE INFANTS

I WANT to show you this small baby today not to bring out especially the physical characteristics of premature infants, but rather to talk to you upon their care and feeding.

This baby, Stella M., was brought into the hospital on September 30, 1922, when one day old. The pregnancy was of something more than six months' duration. On entrance she weighed 1 pound, 13½ ounces. While on entrance her temperature was 100° F., within a few minutes afterward it had dropped to 95° F., and it was only by energetic measures that we managed to keep it somewhere near normal. You will note from the chart that the temperature was quite irregular for many weeks, showing a marked variation between the subnormal temperatures and rises up to 100° or 101° F.

Her weight curve shows the initial drop, the lowest weight occurring on the seventh day after entrance, when she had dropped to 1 pound, 10½ ounces. After a period of four days, when the weight remained stationary, she showed a steady gain, so that at this time (December 5, 1922) she weighs 3 pounds, 13 ounces. This gain has been a steady and consistent one since the twelfth day of life. She had regained her birth weight when she was nineteen days old, and from then on, as I said, showed steady gain.

What has been the method of treatment in this case and what, in general, are the things to be looked for in the premature infant? You will note, first, that the temperature has shown marked variation. In other words, this infant was not able to regulate its body temperature as a normal infant should. Unless this child was supplied with external heat it would not be

able to maintain its body temperature and would certainly not live. Not only is this true, but unless the body temperature is maintained the child does not get the full benefit from the food which it takes and, as a consequence, does not gain weight. This can be very simply and readily shown. A baby which has been gaining regularly on a given formula in the incubator when

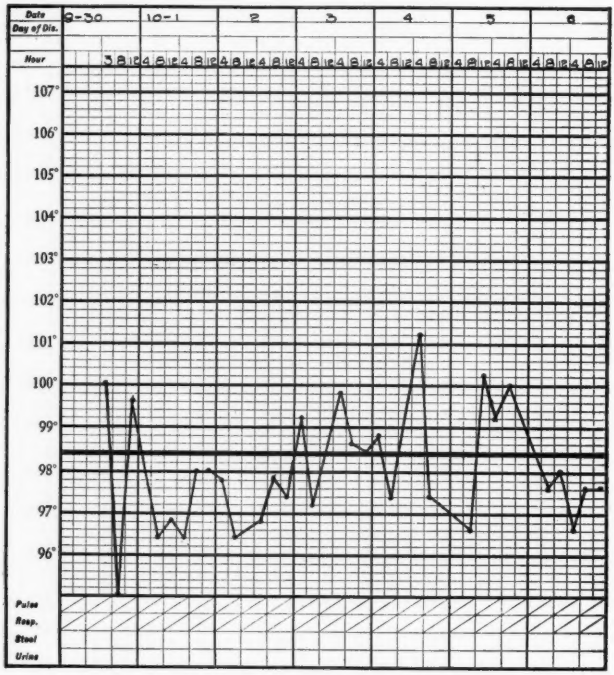

Fig. 80.—Entrance temperature and febrile curve for the following week.

taken out of the incubator for twenty-four hours will frequently show inability to maintain its body temperature, and with this invariably comes either a stationary weight or a loss. We find, therefore, that for the successful treatment of these children it is necessary to apply external heat. How is this best accomplished? It really seems to me that it makes very little dif-

ference how this is done, providing the temperature of the atmosphere in which the child lives is high enough. Under ordinary circumstances this can only be maintained in a properly regulated incubator. One should, so far as possible, prevent rapid changes of temperature, so that one of the prime requisites for a good incubator is that the heat be maintained at a steady

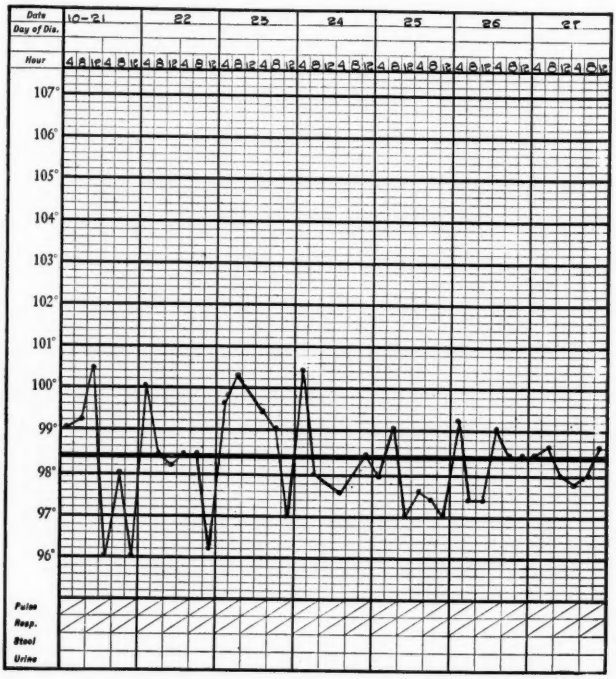

Fig. 81.—Temperature curve showing gradual adjustment of infant's temperature to external conditions.

temperature. For this purpose perhaps the Hess incubator is as well fitted as any which we have. It is simple, can be transported, and can be used wherever an electric current is obtainable. Such current is so generally in use now that this provision does not in any appreciable way detract from the value of this type of incubator. The heat is not likely to suffer rapid changes

because it is conveyed to the incubator not directly from the electric pad, but through the medium of the water in the double-jacketed container. We have other incubators, but none seem to have given us quite as good returns as this.

You will notice when you look at the temperature curve of this infant that frequently the temperature has risen well above

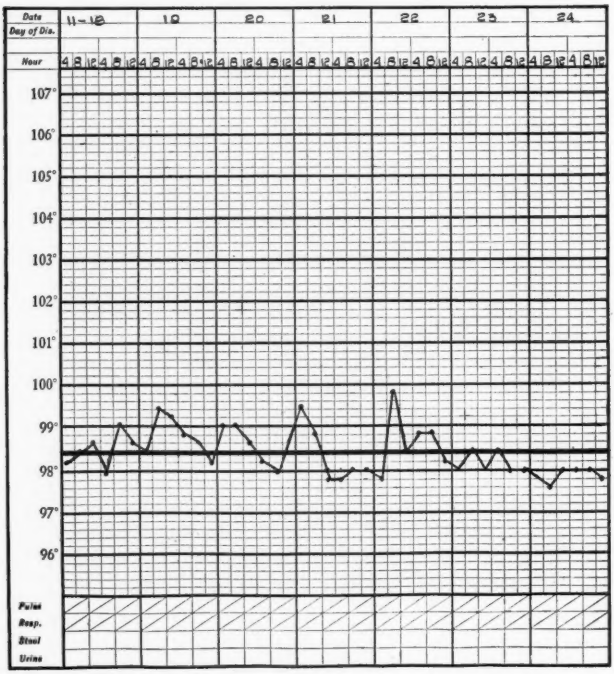

Fig. 82.—Temperature curve after being taken from the incubator.

normal, even as high as 101° F. or higher, and you probably wonder if this represents an infectious process. It does not. These very small infants must be kept, at first, at a temperature of from 95° to 98° F. At times a few degrees one way or the other in the change of this temperature produces a febrile temperature in the infant. So far as I have been able to determine

a febrile temperature produced in this way has had no ill effects, and I have come to disregard it entirely in calculating the dangers to the premature infant.

After a few days with the incubator at 95° to 98° F., we usually try lowering the temperature slightly, a few degrees at a time. The rate at which this is carried out depends altogether on the reaction of the infant; not so much on whether it has a temperature above normal, as to how well, with the change of incubator temperature, it is able to maintain its body heat above 98° F. Finally there comes a time when the temperature in the incubator has been reduced to 80° to 85° F. When that time comes we try placing the child in a warm room to see what the effect will be. If it maintains its body heat it is then left in this warm room for further treatment. The length of time it is necessary to keep an infant in the incubator varies greatly with the individual infant. Of course, in a general way it may be said that it is necessary to maintain these precautions a much shorter time in the larger infants than in the smaller ones, but one can give no definite rule as to how long one may expect to use the incubator in the given instances.

A second consideration is the question of what clothing these babies shall wear. A great deal has been written concerning garments for infants and also regarding the use of specially padded wrappings for prematures. To my way of thinking these are not necessary, and I question very much if they are desirable. While such wrappings do aid in maintaining body heat we may just as easily maintain body heat by the use of properly regulated incubators, and such clothing does materially impede any motion which these children may wish to make. It is true that they usually lie very quietly, but this quiet may be disturbed at any time by a slight movement, and such movements, in my opinion, should not be impeded. We therefore are in the habit of putting on the ordinary garments used in the ward. Perhaps in the first few days these children are wrapped in a little cotton, but this only lasts a short time and is used in only the extremely small babies.

We come now to the question of the nursing of these infants.

This, it seems to me, is a most important question and one which can only be worked out with a lot of experience. The method which we have been following in this clinic has brought a fair measure of success and, I think, may be recommended to you for trial. There are three points to be considered: First, How often will we feed these infants? Second, How will we feed them? Third, What will we feed them?

We must realize that the key to success in the care and feeding of premature infants lies, very largely, in our ability to conserve the energy and powers of these children to the extreme limit. We must, therefore, care for and feed them in such a way that we will protect them from unnecessary handling and unnecessary work in every way. Other things being equal, it has seemed to me that the ideal length of interval for feeding these infants is the four-hour interval. If these children can be given sufficient food, and I think they can, then the four-hour interval means that they are disturbed much less frequently than if fed oftener. This is certainly an important point and one which has not been sufficiently considered in the care of these infants. We therefore used these considerations and adopted the four-hour interval, and feel well satisfied with the results which we have obtained. It should be remembered, however, that this has only been adopted as part of the general scheme and that all the different parts of this scheme must be used at the same time if you are to judge of the results of adopting the four-hour interval.

Next the question comes up as to how we shall feed these infants. Most of them are either too weak or else their nervous systems are insufficiently developed to allow them to make the complicated movements which are necessary to produce proper suckling. It is usually necessary, therefore, to adopt some other means of feeding. Feeding with a spoon or with a medicine-dropper has frequently been resorted to, with a certain measure of success, but we have found that in our cases we were best able to produce a steady gain in weight with the use of gavage. This, when done only every four hours, certainly disturbs the premature infant very little and insures to it the maximum use

of the food which it obtains. Whatever hypothetic reason we may give for this it may be practically proved, for where we have tried to feed the same amount of food with a medicine-dropper, given under exactly the same conditions, almost invariably we find that these children show a stationary weight or even a loss when the change is made. The disadvantage of tube feeding is that it requires a certain amount of refinement of technic to carry it out. In other words, it is frequently necessary that it be done either by a trained nurse or that the children be in a hospital where they can obtain sufficient expert nursing care. Another objection is that when we get ready to quit tube feeding the child must be taught to take the bottle, and this often produces a temporary cessation in weight gain. When, however, we attempt this by replacing one feeding at a time with medicine-dropper feeding or with feedings direct from the bottle we find that the disturbance is not great. I feel quite sure that we have been able to maintain the life of many of these children only as the result of giving the food with the tube.

The next question to be answered is, What food shall we give and in what quantity? These two things must be answered together because the quantity given very palpably depends on the composition. This child was started with albumin milk. The formula to begin with consisted of 3 ounces of albumin milk and $\frac{1}{8}$ ounce of dextrimaltose, six feedings of $\frac{1}{2}$ ounce each. On the following day the albumin milk quantity remained the same and the dextrimaltose was increased to $\frac{1}{4}$ ounce. On the next day the formula consisted of $3\frac{3}{4}$ ounces albumin milk and $\frac{3}{8}$ ounce of dextrimaltose, six feedings of $\frac{5}{8}$ ounce each. On the next day the food was still further increased to $4\frac{1}{2}$ ounces albumin milk, $\frac{1}{2}$ ounce dextrimaltose, six feedings of $\frac{3}{4}$ ounce each. After this time we began to get a little breast milk, so that by October 21st, or when the child was twenty-three days old, it was getting six feedings of $1\frac{1}{4}$ ounces each; three of which consisted of breast milk and three were taken from the following formula: Albumin milk $3\frac{3}{4}$ ounces, dextrimaltose $\frac{1}{4}$ ounce, three feedings of $1\frac{1}{4}$ ounce each. The quantity of breast milk did not increase rapidly, so that at present the child is getting but one feeding of 2 ounces

of breast milk and five feedings consisting of 10 ounces of albumin milk and ¾ ounce of dextrimaltose, each feeding consisting of 2 ounces. You will notice that on the smaller quantities of food the child did not gain weight; in fact, for the first few days, as was to be expected, it lost, but after that the weight remained stationary (Fig. 83). It was only when the food had been raised to a caloric value per pound weight of between 70 and 80 calories that the gain in weight began, and unless the caloric value of the food was maintained at between 75 and 80 this gain did not continue. Such a quantity of food did not produce any untoward gastro-intestinal symptoms in this infant, and it seemed that for this infant such a caloric value was necessary. One cannot, however, generalize from this experience because some infants

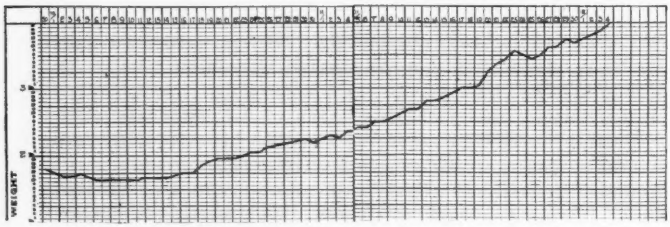

Fig. 83.—Weight curve of Stella M.

will show a steady gain in weight with less food, and others, when the food is pushed to this point, will show gastro-intestinal disturbance. The only rule to go by is to start low and work up gradually. One must remember that any gastro-intestinal disturbance in such an infant is oftentimes followed by disastrous results.

There is one thing more that we must be careful of in the care of these infants, and that is, to prevent respiratory infections. This can be done by keeping away from the infants any attendants who have infections of the upper respiratory tract, and also to see that so far as possible the air which is supplied these infants for breathing purposes is fresh.

Just a few words about the general care. I have already said

something about the clothing. For the first few days these children are oiled, not bathed in water, nor are they kept out of the incubator for any length of time. So far as possible they are changed in the incubator, and the orders are that they shall be disturbed as little as is absolutely necessary.

Water is not given between feedings for the first few days, nor have we found that this in any way detracts from the favorable results.

In closing this subject I would like to call your attention to certain essential features in the treatment of these babies. The cardinal principle of all treatment should consist in conserving their energy to the ultimate limit. Everything should be sacrificed to that. The body temperature must be maintained, nor is it of the greatest importance how this is done. While I have described to you a rather complicated and ideal method for this purpose, other methods will work, and do work, but they are not, in my opinion, likely to bring results in such an extreme case as the present one.

The feedings should, I think, be given at four-hour intervals, providing in the beginning they may be given by gavage. Gradually teach the infant to take the bottle and gradually lower the temperature and decrease the efforts at sustaining bodily heat until you find that the infant, without unfavorable symptoms, can maintain itself both as to its body temperature and as to the taking of milk from the breast or bottle.

Protect this child especially from respiratory infections. One should remember that the mortality among prematures is enormous. Over 90 per cent. of this mortality occurs in the first week of life, and the vast majority of these infants die in the first twenty-four hours. We, therefore, cannot gage results by these infants who die in the first week, and I am sure you will find some help in the care of prematures if you will follow the principles which I have laid down and carry out as well as possible the ideas which I have presented.

CLINIC OF DRS. SOLOMON STROUSE AND PHIL A. DALY

MICHAEL REESE HOSPITAL

THE INTERNIST AND THE SURGICAL ABDOMEN

THE acute surgical abdomen forms an excellent subject for a medical clinic. Almost all patients who require surgical interference for an acute abdominal condition are seen first by the internist or the general practitioner, and it is he who must make the decision which sends the patient to the operating-room. He should also, we believe, make an effort at preoperative diagnosis of the condition, and should not be content with mere recognition of a surgical condition. The diagnostic training received in attempting an anatomic diagnosis which can be checked by biopsy, on the operating-table, will often be of the greatest aid in some later case when difficulty may be experienced in the mere decision as to whether the condition demands surgical interference. Furthermore, nothing is so impressive as one's mistakes, and there is no better method of exposing mistakes than the surgeon's knife and the opened abdomen. When one stops a moment to consider the difficulties of making an exact diagnosis in a chronic condition involving the right upper abdomen, it is quite natural that the latitude of error in an acute abdomen should be great.

Despite the tremendous advances which have been made in the diagnosis and treatment of surgical conditions in the abdomen, the subject is still far from being closed, and there are still too many surgical abdomens allowed to wait beyond the danger point for surgical interference. Surgeons cannot be blamed for this state of affairs; it is up to the man who first sees the patient to recognize the trouble and recommend the

treatment. A ruptured appendix may mean unusual difficulties in diagnosis; it is more apt to mean carelessness on the part of the patient, or delay on the part of the practitioner.

On one point in particular we wish to lay especial emphasis, the relation of food and so-called food poisoning to the surgical abdomen. Having an especial interest in food in relation to health and disease, we have been steadily on the lookout for cases of food poisoning. So far we have failed to find a single example of an *individual* being made sick by food eaten by a *group* of persons unless others in the group were also made ill. It is, of course, a fact that groups of persons can be made sick by poisoned food, but even such occurrences are astoundingly rare, and when an individual complains of abdominal disturbances from food it is most probable that something is wrong with the individual rather than with the food. So-called "ptomaine poisoning" is an entire misnomer; authorities on food now agree that the word and the hazy unknown thing it stands for should be stricken from the medical dictionary and from the minds of medical men. The lives it has cost are not lives sacrificed to food poisoning, but to undiagnosed appendiceal colic or other acute abdominal conditions; yet even today physicians and nurses with stomachache are almost as prone as are patients to look to some article eaten for the cause of the trouble. So a dose of castor oil is taken, the trouble gets worse, and in twenty-four to forty-eight hours there is a dawning realization that perhaps it is something else after all. To illustrate this point: Two nurses were living in the same rooms. One was office assistant to an internist; the other was operating-room supervisor in a big hospital. Both ate some perfectly good-tasting and good-smelling fruit canned by the supervisor's mother; two hours later the supervisor had a pain in her abdomen, was nauseated, and began to vomit. Both girls agreed on the diagnosis: the canned fruit was bad, and a doctor was not summoned until twelve hours later, when he was asked to call whenever he found it convenient. A perfectly frank case of appendicitis was seen, and in a very short time a perfect specimen of an acute suppurative appendix was removed. Such in-

stances are common enough, and as long as physicians are willing to place the blame for "belly-aches" on patients' statements about food without thorough investigation of the belly, so long will the ruptured appendix be seen at the operating-table.

But mistakes in abdominal diagnosis are made for many other reasons, even after most painstaking study. We are, therefore, going to present to you today a collection of patients and of case histories each of which illustrate some diagnostic difficulty.

Case I (History Abstract).—This patient was seen in the early days of practice of one of us (S. S.). He was a young man in the thirties, who walked into the hospital admitting room complaining of abdominal pain, inability to move the bowels, and vomiting. He could speak no English, and nothing further could be obtained in the history. While being examined he vomited large amounts of brown fluid substance which had a fecal odor. His abdomen was rigid, the chest negative. A diagnosis of intestinal obstruction was made and concurred in by a surgical colleague. The patient was immediately taken to the operating-room, the abdomen opened, and no cause for the obstruction found. After a few stormy days without a diagnosis some one who could speak the man's language appeared, and then further history was obtainable. The patient was a plumber and worked a great deal with lead. However, there was no lead-line on the gums, but a careful examination of the blood showed characteristic basophilic granulation. The diagnosis of lead-poisoning was made, proper treatment instituted, and the patient recovered.

Comment.—Although this record is incomplete, the case illustrates a most important element in diagnosing the acute abdomen, namely, the history. Many details of the case are not recalled, and the record is no longer available; what stands out is a picture of intestinal obstruction with fecal vomiting. No history was obtainable, and a hasty operation was performed on objective evidence which seemed incontrovertible.

Case II.—This patient is a man aged thirty years, whose complaint was severe abdominal pain, nausea, frequency of urination, pain upon urination, and fever.

The pain began so suddenly that the patient could remember almost the exact minute. It was sharp, severe, experienced more on the right than left side of abdomen, radiating to the tip of the penis and to the perineum. Urination was less frequent than desire for micturition, because of the fear of pain which the act occasioned. The man felt somewhat nauseated, but did not vomit. He had no chill, but felt extremely feverish.

Questioning as to past history elicits the information that in December, 1920 (nearly two years previously) he had his first attack of abdominal pain, sharp, severe, stabbing pain, felt chiefly in upper right quadrant, and associated at times with burning pain upon urination. These attacks occurred every one to three weeks for some months and varied in duration, from hours to days. One attack was diagnosed as renal colic, but the appearance of jaundice, while under observation, caused the diagnosis to be changed to one of gall-stone colic. In July, 1921 a somewhat similar attack was called acute gastritis, the only positive laboratory findings being a few white blood-cells in the urine.

The patient was first seen by us twenty-four hours after the onset of his present attack. He had had medical attention and several hypodermics which gave him but little relief. His pain was very severe and an interesting feature of it was the regularity of the paroxysms which lasted two to three minutes with marked relief during intervals of like duration between paroxysms.

Temperature was 102° F. by mouth, 105 rectally; pulse 120, respirations 28 and entirely thoracic. Throat, heart, and lungs were negative. Abdomen was rigid—a board-like rigidity—and exquisitely tender, more pronounced on the right, but not confined to the right side.

His white blood-count was 17,000, with more than 90 per cent. of polymorphonuclear leukocytes. Urine showed a specific gravity of 1018, a plus albumin, many granular and hyaline

casts, white blood-cells, and red blood-cells. Blood-pressure 128 systolic and 78 diastolic.

In spite of morphin the severe pain and board-like rigidity of the abdomen persisted, and after eight hours the consulting surgeon made a diagnosis of a ruptured viscus and immediately performed a laparotomy. Beginning with an incision in the upper right abdomen the gall-bladder and stomach were explored and found negative. There appeared, however, a small amount of pus welling from the lower abdomen. A right rectus incision in the lower quadrant revealed the source of the trouble in a purulent peritonitis with fecoliths scattered among the loops of intestines, a ruptured appendiceal abscess, and a gangrenous appendix.

A somewhat stormy postoperative course resulted eventually in recovery.

Comment.—This patient when first seen was considered to have renal colic. His quite characteristic history of previous attacks, the very definiteness of the history of the present pain shooting to the tip of the penis, the difficulty of urination influenced us too much, and hid the importance of the physical findings of peritonitis, the board-like rigidity, and the severe paroxysmal pains. In this respect the case is the antithesis of the first case in which absence of history prevented a correct diagnosis. In the present case too little attention to an evident marked disagreement between history and physical findings delayed the diagnosis. Although the story sounded like a kidney colic, the findings were not consistent with the story; and as the findings are objective and the story subjective the proportion of attention to the two was not well balanced.

Case III.—This patient is forty-eight years of age. Before the onset of the present trouble he had been under our observation for several years for somewhat vague disturbances—nausea and distention—after eating. He is a traveling salesman, and despite our earnest requests for an opportunity to study him long enough to make a diagnosis, he has always refused. Occasionally he visited the office for a physical examination, which

invariably revealed a perfectly healthy man, with no tenderness, rigidity, or abnormal abdominal condition. At 3 o'clock on the afternoon of the onset of the present illness he visited the office because of constipation and the same vague gastric distress; he was again given a thorough physical examination. The abdomen was everywhere soft, there was no tender spot, and no masses. He left the office and went home.

At 6 P. M. he was suddenly seized with an excruciating pain in the epigastrium, nausea, and vomiting. The pain radiated to the lower abdomen. The patient had not eaten since breakfast, and the vomitus consisted of clear fluid. He noticed that after the pain the abdomen became stiff. He had no chill and no fever. A neighborhood physician was called; he administered morphin and left. The pain eased only slightly and another physician was summoned. At 8 P. M. we were called to see the patient.

Physical Examination.—The patient was having paroxysms of pain in the midepigastrium. He, however, did not look ill. His temperature was 98.6° F., pulse 52, respirations 20. Except for the abdomen the general examination was negative. Both recti were rigid, the right more than the left. Tenderness was general, most marked in the right hypochondrium.

A diagnosis of ruptured gastric ulcer was made and the patient immediately sent to the hospital. There the white blood-count was 17,000. Urine showed specific gravity of 1026, trace of albumin, positive acetone, and microscopically a few granular casts and white blood-cells.

By the time he was settled in the hospital at 10 P. M. he was still having severe pain, but altogether in the right hypochondrium. Temperature, pulse, and respirations remained normal. The abdomen remained rigid, but there was definitely more tenderness and rigidity in the right hypochondrium than elsewhere. This localization of signs in addition to the complete absence of increased temperature (both by mouth and by rectum), of change in the pulse-rate, or of general signs of intoxication made us doubt the original diagnosis of ruptured ulcer, and suspect we were dealing with a pathologic gall-bladder. We

watched the patient all night. There was at 8 A. M. still no change in the patient's general condition, the temperature had risen to 99.2° F., the pulse was 72. Dr. McArthur then saw the patient, concurred in the original diagnosis of ruptured ulcer, performed an immediate laparotomy, and found a perforation of an ulcer in the first portion of the duodenum. The abdomen was filled with clear fluid and there was no peritoneal reaction. The patient made an uneventful recovery, showing no increase in temperature or elevation of pulse-rate at any time.

Comment.—The delay in calling a surgical consultation in this case should have been avoided. The atypical history, the shifting of signs to the gall-bladder region, and the absence of any signs of reaction to a peritoneal insult were responsible for the delay. The explanation of the last-named phenomenon probably lies in the fact that cultures from the peritoneal cavity taken at operation were sterile, due probably to the absence of any food in the stomach at the time of perforation and the presence of a gastric juice of high acidity.

Case IV.—This man, who is fifty-two years of age, has a past history very similar to that of the preceding patient: distention and nausea immediately after eating. This combination has been present at irregular intervals for three or four years. Alkalies afforded no relief, but catharsis helped greatly. Physical examination including Roentgen ray showed no cause for the trouble. His attacks have been less frequent since a tooth extraction nine months ago. He had an attack two weeks ago, and the present one began twenty-four hours previous to entrance into the hospital. The attack began with a feeling of pressure in the epigastrium which developed into cramps. The customary relief from catharsis was not forthcoming. He had some nausea, but no vomiting. Headache was rather annoying and he was conscious of a moderate tenderness of the abdomen.

The only physical findings eighteen hours after onset were tenderness in the right lower quadrant and definite muscular resistance at the same site. Temperature 99.8° F. rectally, pulse 80, and respirations 18.

Urine showed a specific gravity of 1025, a trace of albumin, and both red and white blood-cells microscopically.

The blood examination revealed a hemoglobin of 80 per cent., red blood-cells 4,320,000, white blood-cells 16,600, of which 84 per cent. were polymorphonuclears. Hemoglobin and white corpuscle count remained about constant, there being a slight decrease in the leukocytes, followed some hours later by an equal increase. The temperature and pulse remained the same.

Operation was performed about twenty-four hours after patient was first seen or about forty-eight hours after onset. A purulent appendix with a necrotic tip lying on the brim of the pelvis was removed, rupturing during removal. The pathologic report was acute diffuse suppurative appendicitis, with necrosis of the tip of appendix. Recovery was somewhat slow, but complete.

Comment.—A very nervous and introspective man for years subject to vague abdominal distress similar to the symptoms complained of by the patient last shown. During this period he was examined by numerous physicians and had several complete Roentgen-ray examinations, without a successful localization of the source of his trouble. An attack similar to preceding ones occurs, associated with the findings of an acute appendicitis, but with red and white blood-cells in the urine. This brings up two separate points for discussion. At operation a purulent appendicitis was found, the necrotic tip of the appendix lying on the pelvic brim, and undoubtedly causing the presence of the inflammatory cells in the urine. The presence of blood- and pus-cells in the urine of patients with acute appendicitis has recently been noted by several observers; in this case the definite history and physical signs of acute appendicitis warranted the diagnosis despite the urinary changes.

The relation of the present attack of acute appendicitis to the long previous history of gastro-intestinal disturbances is problematic, as the operation is still too recent to permit conclusions. It is probably true that most indefinite stories, such as this patient's, are caused by definite lesions in some one organ, even when the best of modern medical diagnostic methods can-

not locate the lesion. Straight neuroses do occur, but they usually can be made to yield to proper psychotherapeutic measures; persistent gastro-intestinal disturbances even in a neurotic individual ultimately will be shown to have a basis in some organ.

Case V.—The next patient is a married woman, thirty-five years of age, who twenty days previous to date of first observation had a menstrual period which she considered normal. Two days later she experienced crampy pains in the abdomen, nausea, and "spotting," she felt chilly, but had no fever. For four or five days she continued to have slight pain in the lower abdomen and she remained in bed for seven days, following which, while up and around, she had some slight abdominal distress. She saw a number of doctors during the week, various diagnoses being made. Four days previous to admission she experienced chills, severe abdominal pain, more pronounced on right side and radiating to the right shoulder, vomiting only twice.

On the day of admission to the hospital (November 2, 1921) she consulted an eminent obstetrician and gynecologist, who pronounced the pelvis negative and referred her to an internist. On her way to the office in a cab she fainted. She was brought into the office in a condition of shock, and after a hurried examination, which revealed a very tender abdomen, especially the right lower quadrant (no rigidity), she was sent immediately to the hospital.

Examination revealed the physical findings as above recorded, and, in addition, temperature of 100.4° F., pulse 120, and respirations 32; hemoglobin 80 per cent., red blood-cells 3,610,000, white blood-cells 27,000, with 95 per cent. of polymorphonuclears. The urine was normal.

She immediately began to improve clinically, temperature remained about 99, pulse 100, and respirations 20. The leukocyte count dropped to 7000. She had some epigastric pain and some nausea. Some vaginal bleeding was present which, unfortunately, was considered a menstrual flow that had begun a few days early.

This picture remained unchanged for several days, a diagnosis of acute appendicitis and cholecystitis being made. She was operated on November 9th, and a ruptured ectopic pregnancy found. The right tube had contained the fetus, a small oval mass 4.5 x 3 cm. being recovered. Chorionic villi and decidual tissue were present in the tube. A right salpingectomy and an appendectomy were performed. The pathologic diagnosis was ruptured tubal pregnancy and chronic obliterative appendicitis. Recovery was uneventful.

Comment.—This illustrates a case in which one could hardly fail to consider a ruptured ectopic, yet the idea was not very seriously entertained because of an opinion by a very good obstetrician that the pelvis was negative. In spite of the fact that diagnoses of early ectopic pregnancies are seldom made until rupture occurs, it must be admitted that the negative report influenced succeeding opinions far more than should have been the case. In this particular instance the combination of menstrual disturbance and acute abdominal pain followed by shock should have made the diagnosis despite the negative pelvis.

SUMMARY

These few cases, each chosen to illustrate some difficulty in diagnosis of the acute abdomen, are shown to emphasize to the general practitioner or internist the fact that his is the responsibility of early diagnosis and early treatment of this group of cases in which delay or error may mean a preventable death. Perhaps the most important lesson we have learned from a study of our patients is that every acute abdominal disturbance may potentially be a surgical condition. Food rarely causes acute abdominal trouble unless something is wrong with the person eating the food or unless groups of individuals become sick after eating the same food. In doubtful abdominal conditions to diagnose ptomaine poisoning or to treat by morphin injection is unpardonable. Many acute abdominal conditions present at onset vague syndromes, which is the best reason for deferring "medical" treatment, and for watchfully waiting for the change which makes diagnosis possible. And, in conclusion, if we

accept the dictum that every acute abdominal condition may be a surgical condition, it follows that in all such cases the surgeon should be called in early. While we firmly believe that the medical man should try to make an anatomic and physiologic diagnosis, he should not jeopardize his patient's life by delay in conferring with his surgical colleague during the period of observation.

CLINIC OF DR. JULIUS H. HESS

COOK COUNTY HOSPITAL

SOME CHRONIC ABDOMINAL CONDITIONS IN INFANCY AND CHILDHOOD. PRESENTATION OF THREE GROUPS OF CHRONIC ABDOMINAL CASES

I. Stenosis of the Lower Esophagus at the Cardia.
II. Partial Imperforate Anus.
III. Dilatation and Hypertrophy of the Colon:
1. **Hirschsprung's Disease. Primary Dilatation of the Colon.**
2. **Celiac Disease, Intestinal Atrophy and Dilatation, Chronic Digestive Insufficiency.**
3. **Rickets with Secondary Dilatation of Colon.**

I. STENOSIS OF THE LOWER ESOPHAGUS AT THE CARDIA—GASTROSTOMY WITH DILATATION OF THE STENOSED PORTION

Case I.—Arthur S., aged twenty-three months, entered Michael Reese Hospital January 18, 1921, and was discharged February 28, 1921. He therefore appears before you two years following his operation for the relief of the obstruction. The history of the case as given by his mother in 1921 was as follows:

Normal birth, weight 5 pounds, 4 ounces; from the age of two months on baby would vomit soon after nursing (this would occur just after nursing or within thirty minutes); it was constipated from onset; vomiting was not projectile. Baby remained on breast entirely until seven months old, at which time the weight was about 9 pounds; vomiting continued and was aggravated by artificial feeding (which was started after the seventh month). When one year of age vomiting had assumed the projectile type; baby had pneumonia at eleven and fourteen months; weighed 14 pounds at twenty months.

Operation.—Gastrostomy performed by Dr. Alfred A. Strauss. After opening the abdomen palpation revealed a definite thickening around the cardiac end of the stomach and adhesions between the lower end of the esophagus and diaphragm. There was also a large Meckel's diverticulum. An incision was made in the anterior wall of the stomach and the cardiac portion was explored. A constriction at this point prevented the finger from entering the esophagus from below. The esophageal wall was

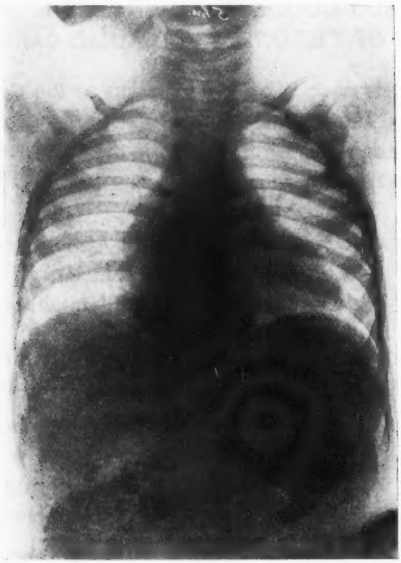

Fig. 84.—Arthur S. Stenosis of the lower esophagus at the cardia.

thickened and the lower end of the esophagus greatly dilated. An artery forceps was passed in from below and the cardia was dilated. A large catheter was passed through the mouth into the esophagus and pulled into the stomach, after which the stomach was closed by three layers of sutures. The catheter was left *in situ* for eight hours.

Subsequent Course.—Following the operation the infant developed a generalized bronchopneumonia from which it made

a very slow recovery, leaving the hospital on the thirty-ninth day after operation in good condition.

Subsequent History.—Two years later the child is able to eat solid diet without inconvenience and has developed normally.

Case II.—Francis E., aged three years, entered Michael Reese Hospital 7/7/21, died 7/21/21. This child died on the third day following gastrostomy for the relief of the obstruction and I am presenting the roentgenograms and history. As in Case I, the diagnosis was not made until a short time before operation.

History.—She entered the hospital with a diagnosis of cardiospasm and hypertrophic pyloric stenosis. The delivery was spontaneous and the infant was fed on the breast for four months, following which she was bottle fed, with added cereals. Talked at one year and walked at fourteen months.

Present Complaint.—At nine months the infant commenced to vomit, and this occurred practically after each meal. The vomiting followed shortly after taking food and at times before the meal was finished. Between meals she frequently vomited varying quantities of clear mucus-like material. Before vomiting she frequently had a wheezing type of respiration which was relieved by vomiting. Her symptoms have become more aggravated during the past year. The child eats cereals and other soft foods, but refuses solid foods. Bowel movements are regular and of fair size.

Past Illnesses.—Measles at two and a half years, whooping-cough at one and a half years. Family history is negative. One other child living and well one year old. No miscarriages, no history of lues, tuberculosis, or malignancy obtainable.

Physical Examination.—Reveals a well-developed and well-nourished child who is apparently not acutely ill. Head, scalp, eyes, ears, mouth, nose, neck, chest, lungs, and heart are negative.

Abdomen: No distention, rigidity, or tenderness present; no peristaltic waves visible or audible; no tumor masses palpable. Liver, spleen, or kidneys are not palpable. Genitalia, extremities, and reflexes are negative.

The urine is negative. Blood examination: Hemoglobin 65 per cent.; red blood-corpuscles 3,390,000; white blood-corpuscles 8400; neutrophils 49; small mononuclears 30; large mononuclears 18; transitionals 2; basophils 1. Von Pirquet was negative.

Course in Hospital.—Patient vomited after every meal, both immediately and sometimes several hours afterward. Vomiting not projectile in type. Vomitus composed largely of saliva, mucus, and some food.

Fig. 85.—Francis E. Stenosis of the lower esophagus at the cardia, and hypertrophic stenosis of the pylorus.

Fluoroscopic Examination.—Shows obstruction at the cardiac and pyloric portions of the stomach with a dilatation of the esophagus. Barium meal passes into the esophagus and a point of obstruction is noted as the esophagus passes through the diaphragm. The stenosis is not complete. The barium is seen to slowly dribble through into the stomach. The esophagus is dilated above the obstructed point. At no time did the spastic

stricture open. The stomach is orthotonic, empties in three hours. No filling defects. Duodenal bulb not visualized. The barium meal is seen in the ilium and enters into the cecum in the five-hour observation. The outline of the esophagus is smooth and regular. Fusiform in shape.

Operation.—Patient was operated by Dr. A. A. Strauss. A midline incision was made about 6 cm. long (Figs. 21, 22), extending from the costal cartilage down. The stomach was

Fig. 86.—Francis E. Stenosis of the lower esophagus at the cardia and hypertrophic stenosis of the pylorus.

delivered, the thickened pyloric sphincter was split in the usual way. The stomach was then opened, aspirated, and the cardiac opening examined. It was found to be about the size of a match. A soft catheter was passed through the mouth, but it would coil up in the dilated esophagus and would not pass into the stomach. A harder catheter was then passed from the stomach into the esophagus and out of the mouth. An 8-inch hemostat was snapped on the lower end of the catheter and pulled through

the stenosed esophagus. The stenosed portion was then dilated. A few adhesions were found between the esophagus and the diaphragm. These were broken up. The abdomen was then closed in layers in the usual way.

Postoperative Course.—The child suffered from severe shock following the operation and developed a generalized bronchopneumonia, dying on the third day following the operation.

Summary.—To all appearances this infant remained without symptoms of esophageal stenosis during the first nine months of life, following which the condition became gradually worse up to the time of its entrance into the hospital at the age of three years. At this time the child was quite well nourished, and although it vomited very large quantities consisting of a considerable part of its diet and mixed with saliva, and mucus, it retained sufficient food to allow it to develop in a fairly normal manner. The outstanding factor in arriving at a diagnosis before roentgenologic examination was the vomiting of saliva and mucus. The etiologic factors concerned in the development of the stenosis remain in doubt.

Summarizing the postoperative course of these 2 cases of stenosis of the esophagus, the tendency toward the development of pulmonary complications is most striking. Whether this was due to irritation of the diaphragm and surrounding tissues or secondary to aspiration is questionable. One of the cases resulted in a complete recovery and normal functions, the second in death.

II. PARTIAL IMPERFORATE ANUS

Case I.—Harriet F., aged six months, admitted February 8, 1923 and discharged February 25, 1923.

Present Complaint.—Frequent small ribbon-like bowel movements, the movements followed marked straining associated with tenesmus.

Onset and Course.—The infant has been having frequent bowel movements since two weeks of age, always formed, but of small diameter and frequently mixed with masses of mucus. Usually six or eight movements a day. Tenesmus and pain were first noted when the infant was three weeks of age; the condition has

gradually grown worse. At times the infant vomits, but this has been absent for over two months.

Personal History.—Full-term normal delivery; birth weight 5 pounds, 14 ounces. Mixed feeding since birth. Family history negative.

Past Illnesses.—Has been seen by a number of physicians, who have treated it as acute intestinal indigestion and colitis.

Physical Examination.—Well-nourished infant weighing 13 pounds, 9 ounces, who does not look sick, smiles when its attention is attracted, and is seemingly in good health. Skin is elastic and has a fair amount of subcutaneous adipose tissue. She can sit up without support. Head, ears, eyes, nose, mouth, and neck are negative. Chest, heart, and lungs are negative. Abdomen not distended, moderately resistant, and not tender. Liver palpable, spleen and kidneys are not. There are several small hard masses varying from 1 to 3 cm. in diameter located in the left upper quadrant of the abdomen, and several less distinctly palpable about the same size in the lower quadrant. These masses feel hard and at first suggest enlarged isolated lymph-nodes. Upon careful palpation they can be molded. Each time the abdomen is examined the abdominal muscles contract, the child starts to strain, and a small ribbon-like stool is passed, attended by painful tenesmus. Upon examination of the anus with considerable effort the small finger can be passed into the rectum and a large fecal mass can be palpated in the rectum. Passing of the finger results in bleeding from the anus.

Laboratory Findings.—Urine showed alkaline reaction and contained numerous pus-cells. Blood examination showed hemoglobin, 90; red blood-cells, 5,500,000; white blood-cells, 15,000; neutrophils, 48; small mononuclears, 44; large mononuclears, 7; transitionals, 1. Von Pirquet test and throat cultures were negative.

Roentgenologic Examination.—The fluoroscopic examination of the gastro-intestinal tract discloses a normal stomach that empties within a normal time. No obstruction noted in the small intestine. At twenty-four hours the barium is in the colon, some of it in the cecum and ascending colon, and some

of it in the descending. The balance of the colon is empty, but filled with gas. There are no colon defects to be observed. At forty-eight hours the barium is entirely in the descending colon, and at seventy-two hours presents much the same appearance. No defect noted. The emptying time is very slow; the normal colon of this type usually empties at twenty-four hours. Following the bismuth meal there resulted an impaction in the lower rectum which was relieved with considerable difficulty.

Subsequent Course.—The repeated administration of liquid petrolatum caused the intra-abdominal masses to disappear.

Operation.—Patient was operated on the thirteenth day. The anus was incised laterally and dilated with finger under local anesthetic. The infant left the hospital five days later, the mother having been taught to insert the finger into the rectum, which she does once daily. The infant has been seen once a week since leaving the hospital and now has free bowel movement once or twice daily.

The abdominal masses have disappeared and the tenesmus has not recurred in the past three weeks.

Summary.—In the presence of a continuous history dating from the second week after birth of frequent small ribbon-like bowel movements there had been no examinations of the anus or rectum. Of interest are the intra-abdominal masses which disappeared under preoperative treatment. One of the most striking features was the prolonged retention of the opaque meal in the colon even after the rectum had been emptied by the previous treatment. The simplicity of the operation and post-operative care, together with the excellent result, are the most striking features.

Case II.—*Congenital Anomaly of Colon, Partial Imperforate Anus, and Rectovaginal Fistula.*—Ruth D., aged seven months, admitted November 27, 1922 and died December 7, 1922.

I am presenting the history, roentgenograms, and pathologic specimens of this case.

The child entered the hospital for the first time on November 27, 1922, complaining of the following symptoms: Vomiting,

failure to gain weight, abdominal tumor, discharge of feces from the vagina, and a slight fecal discharge from the anus. It was noticed after birth that the anus was imperforated and that feces discharged from the vagina. When two months old it was operated on, at which time the anus was perforated and a probe was passed through the perforation. This was repeated about every five days for six weeks. Subsequently it was found that a small amount of fecal material passed through the anal opening while the major portion continued to pass through the fistula in the vagina. The bowel movements had been peculiar in that there would be a period over several days without bowel movement, and coincident with this there would be a swelling in the lower abdomen. Subsequent to this period of constipation there would be copious and frequent stools. The vomiting had been present almost daily for the last twenty-six days, it is projectile in nature and comes on immediately after feeding. The vomiting seemed to have no relation to the periodic constipation. Frequently it was so severe that at least 75 per cent. of the feedings was not retained. The loss in weight had been rather steady and amounted to 3 pounds in the last twenty-six days. The tumor in the lower abdomen appears during the periods of constipation due to the accumulation of feces, and at these times peristaltic waves are observed. There had never been any signs of a complete intestinal obstruction. The discharge of feces from the vagina had caused a superficial irritation of the external genitalia.

Past History.—There were no other children and no congenital anomalies in the mother or father. The birth weight was $8\frac{3}{4}$ pounds. Its subsequent development was normal, but retarded.

The *physical examination* on November 27th revealed a well-developed and fairly well-nourished infant about seven months old, who did not appear acutely ill. Examination of the head and neck, chest, and heart did not reveal any abnormalities. The abdomen was slightly distended. There was no tenderness or masses palpable. There was some edema of the extremities. There was a moderate fecal discharge from the vagina.

Laboratory Findings.—The urine was essentially negative. The blood showed a white blood-count of 11,200.

The roentgenologic findings on November 29th showed the stomach to be of normal appearance and empties completely in four and a half hours. In twenty-four hours the bismuth was collected in the cecum and a small amount in the terminal ileum.

Fig. 87.—Double transverse and descending colon. The anterior colon s shown as ending in a blind pouch. Leading into this pouch is a tract connecting it with the vagina. The posterior colon, about twice the size of the anterior colon, is connected with the anal opening.

The cecum, ascending and transverse colons were dilated and contained considerable gas. In forty-eight hours the same picture was observed. In passing a catheter through the recto-vaginal fistula barium was forced into the colon and disclosed a normal colon from the splenic flexure down as well as the greater portion of the transverse colon. A catheter was inserted into the rectum and met with an obstruction about 5 cm. from

the skin margin. The baby was discharged on December 7th because it was decided that it would be better to allow the child to regain strength and weight before undertaking an operative procedure. On December 7th at about 10 A. M. the child had convulsions and generalized twitchings. It was sent to the hospital and arrived at 1.30 P. M. It lived only twenty minutes after being admitted. The spinal fluid was under normal pressure and slightly bloody.

Autopsy Findings.—Gastro-intestinal tract (Fig. 87): The stomach appears normal. There is no hypertrophy of the pyloric sphincter. The entire small intestine appears normal, and there are no congenital anomalies of it. The mucosa throughout the small intestine is of normal appearance.

The cecum is dilated and hypertrophied. It measures in its transverse diameter about 5 cm. The mucosa is thickened and is thrown up into small irregular folds. The ascending colon is likewise dilated and hypertrophied. At 14 cm. from the ileocecal valve there is a thin septum which runs directly across the bowel and divides the remaining colon into two distinct channels. The openings from the proximal colon into the two distal colons measure 1 and about 2 cm. in diameter. In examining the remainder of the double bowel, one finds that the *anterior colon* which has the smaller of the openings into the proximal colon is of about normal size throughout its transverse and the upper portion of the descending colon. At 17 cm. from the anus this anterior colon becomes enlarged to a pouch-like dilatation, which measures about 6 cm. in diameter. The wall of this pouch is thickened, fibrous, and is ulcerated superficially over the distance of about 2 cm. At the beginning of this pouch-like dilatation there is a perforation into the posterior colon which measures 0.8 cm. in diameter. This is a clean-cut perforation, the edges of which are somewhat hemorrhagic. Tracing this gut further it is seen to empty into the lower portion of the vagina very near its mucocutaneous junction. *Tracing the posterior colon* from its beginning at the hepatic flexure it is about twice the size of the anterior colon throughout its transverse position, and the descending portion

of this posterior colon is dilated and hypertrophied and measures about 4 cm. in diameter. In the region of the ulceration noted in the anterior colon there is a moderate constriction of this posterior colon with a superficial band-like ulceration of its mucosa. In this region there is seen the opposite surface of the perforation which has occurred through the septal wall between the anterior and posterior colons. Distal to this slight constriction the posterior colon is dilated, thickened and fibrous, and forms a blind pouch which measures 8 cm. in diameter. After removing the organs *in toto* and further examination it is seen that the blind tract which is connected with the anal opening comes in immediate relation with the anterior colon, but there is no distinct opening between this blind pouch and the anterior colon. There is a common septum between the anterior and posterior colon throughout its entire extent.

The upper portion of the vagina appears normal and presents on its posterior wall near the mucocutaneous junction the vaginal opening of the rectovaginal fistula.

III. DILATATION AND HYPERTROPHY OF THE COLON

1. Hirschsprung's Disease. Primary Dilatation of the Colon.—This patient, Joseph M., aged three years, was blue when born, but was resuscitated within a few minutes. He was breast fed until one year old, and then put on a mixed diet, meats not being given until the end of the second year. (Mother had been in labor three days and child was delivered with forceps.) Child was markedly constipated from birth and was given castor oil, olive oil, enemata, etc. When bowels were constipated he would vomit upon the administration of food and water. It was always necessary to use a cathartic. A stupid state accompanied long bowel retentions. He complained of pain in the abdomen and had fever at frequent intervals. Micturition was frequent and there was some loss in weight. Mother stated that the abdomen frequently became very large. The family history was negative.

Physical examination was negative except for the abdomen. There was a large tumor mass about the size of a small orange

just above the pubis and reaching nearly to the umbilicus. It was freely movable. The child was given castor oil and various kinds of enemata, but only with fair results. Rectal examination revealed a mass of feces. After being in the hospital about ten days the mass disappeared.

Fig. 88.—Joseph M. Hirschsprung's disease. Primary idiopathic dilatation of the colon.

Roentgenologic Examination (Fig. 88).—First examination eight hours after ingestion of opaque meal showed hepatic flexure, cecum, ascending colon, and first portion of hepatic flexure well filled out. There were no adhesions or defects. Transverse colon and splenic flexure were much distended with gas. At the end of twenty-four hours the cecum and ascending colon were empty. Opaque meal in descending colon was highly diluted with fluids and the colon dilated. Opaque enema showed that the rectum filled normally. The balance of the colon was

very large and dilated, being full of fluid; the cecum was very large. An enema consisting of 1500 c.c. of barium sulphate and buttermilk was used, but was not sufficient to fill the colon.

Laboratory Findings.—Urine was negative. The blood examination showed 80 per cent. hemoglobin, 4,280,000 red blood-cells, and 9400 white cells, of which 68 were neutrophils, 21 small mononuclears, 10 large mononuclears, and 1 eosinophil.

Subsequent Treatment.—The treatment which we will recommend in this case will be as follows: Mineral oil 1 ounce at bedtime, together with the rectal installation of 2 ounces of cottonseed oil at bedtime. Should this medical treatment be insufficient to promote emptying of the bowel, the mineral oil should be repeated each morning, a like amount being given. In our experience at times it is necessary to give further laxatives, and I have found compound jalap powder in teaspoonful doses a good adjunct to the above treatment. The diet should consist largely of fruits, green vegetables, and carbohydrates. In some of the cases the increased fermentation due to the carbohydrates are valuable because of their tendency to increase peristalsis. In some children, however, a tendency to increased tympanitis may make it necessary to feed a low carbohydrate diet. Lactic acid milk should replace sweet milk. Dilute hydrochloric acid in doses of 15 minims after meals is also administered.

As further medication he will be given 1/320 grain of thyroxin three times daily and 2 grains of desiccated pituitary substance (posterior lobe) once daily. His abdomen will be massaged night and morning and he will wear an abdominal support.

This course of treatment must be considered in the light of palliative measures, and if the results are unsatisfactory, surgical interference should be given proper consideration. Resection of the colon, probably best performed in three steps, is the operation of choice.

2. **Celiac Disease, Intestinal Atrophy and Dilatation, Chronic Digestive Insufficiency.**—This patient, Ralph T., was admitted to the hospital September 19, 1921 at the age of three years. He is now five years of age, and I believe you will agree

with me that he is fairly well developed for his age. The history, as recorded at the time of entrance to the hospital, is as follows:

He was delivered normal; had his first teeth at ten months; sat erect at five months; talked single words at twelve months; walked at fourteen months; breast fed for thirteen months. Mother states that she nursed nearly all the time to keep him quiet. Following weaning the infant was placed on a milk and cereal diet, with no vegetables or fruit, until fifteen months of age.

Present Trouble.—Alternating constipation and diarrhea, distention of abdomen, marked irritability, stationary weight, and intermittent fever. The diarrhea began at fifteen months. Previous to this time the child was markedly constipated. The mother states that he passes large, white stools, containing considerable mucus and at times during the spells of constipation and during the diarrheal attacks undigested food, watery, and mixed with much mucus. The stools are always very foul. The attacks of constipation are usually associated with fever, and the patient also has a temperature during the height of the diarrheal attacks.

Previous Illness.—He has had repeated attacks of tonsillitis; chickenpox at two years; otitis media at two and one-half years; convulsions at different times between the ages of eleven months and two years. The mother thinks these were due to fever and diarrhea.

Family history is unimportant.

Physical Examination.—The child is small in stature, undernourished, pale and anemic in appearance, with marked evidence of rickets. Head is rachitic in appearance. Eyes, ears, nose, throat, and neck are negative. Chest is not well developed, showing evidence of healed rickets. Lungs and heart are negative. Abdomen is very large, protuberant, and tympanitic. There are no areas of rigidity or tenderness. No peristaltic waves are visible and no tumor masses are palpable. Liver extends 5 inches below the costal margin. Genitalia are negative. Extremities are poorly developed and the muscles are flabby.

Laboratory Findings.—Stools are large, bulky, colorless, and mushy in consistency, with a very foul odor, consisting largely of unabsorbed fat in the form of soaps.

Bacteriologic examination shows that colon bacilli predominate. No typhoid, paratyphoid, or dysentery bacilli were found.

Fig. 89.—Ralph T. Celiac disease. Secondary dilatation of the colon with chronic digestive insufficiency.

Protein tests are negative to cow's milk, oats, wheat, potato, and egg yolk, faintly positive to egg-white.

Gastric contents show no hydrochloric acid one hour after feeding.

Roentgenologic Examination (Fig. 89).—Stomach is large, but normal in outline, empties in four hours, at which time the barium was distributed throughout the pelvic ileum and prox-

imal colon. At the end of twenty-four hours the colon is almost empty. Opaque enema shows a marked dilatation of the large intestine beginning at the ileocecal junction. The large bowel is greatly increased in size from the splenic flexure to the rectum.

Summary.—This boy was fed thirteen months at the breast, received no fruit or vegetables until fifteen months of age, was markedly rachitic, and had repeated convulsions from the eleventh month to the end of the second year, in all probability due to spasmophilia. At fifteen months he first developed the stools typifying chronic intestinal indigestion, with alternating constipation and diarrhea. In all probability his early rickets was an early factor in the development of his enlarged colon and sigmoid. The absence of sufficient vitamin-containing foods in his diet is worthy of consideration as an etiologic factor.

Treatment.—In outlining the treatment for the group of cases which may be classified as chronic digestive insufficiencies it should be remembered that the function of practically the entire gastro-intestinal tract is involved, and it is not improbable that the liver and pancreatic function are also disturbed. While the etiologic factors which may produce such a pathologic and clinical picture may differ widely and therefore be of little assistance in outlining the treatment, if the clinical findings are clearly borne in mind definite conclusions as to treatment may be established. Due to the fact that little or no fats can be digested and that the easily fermentable sugars must be held to minimum quantities, the greater part of the diet must of necessity be composed of protein and well-cooked starches. At all times sufficient food must be given to meet the child's energy requirements and at least moderate growth. As a basis for this boy's diet he was at first given skimmed lactic acid milk, of which he received a minimum of 1 quart daily. During the early part of his treatment from 1 to 3 ounces of dried casein was added to his skimmed lactic acid milk. At times he tired of this combination, and instead of the lactic acid milk, skimmed sweet milk was used. Well-cooked farina formed the basis of the carbohydrate in his diet. This was alternated with cream of wheat and oatmeal. In some of our cases the latter has, how-

ever, not been well taken. Rare broiled scraped beef was next added. At first 1 tablespoonful was given daily. A soft-boiled egg was added during the second week of his treatment and at about the same time two small slices of toasted bread. During the third week small quantities of baked potato were alternated with well-cooked spinach, asparagus, vegetable marrow, and stewed celery. Early in the treatment it soon became evident that he craved a further addition of carbohydrates, and 1 to 3 ounces of corn syrup or honey were added to his daily ration. To supply sufficient vitamins orange juice, at first 1 ounce and later 2 ounces daily, and 2 teaspoonfuls of cod-liver oil were administered; 15 drops of dilute hydrochloric acid was added to the orange juice twice daily, and as further medication he received citrate of iron and ammonia, 5 grains, twice daily. During his periods of constipation he received an injection of 2 ounces of cottonseed oil at bedtime, which he was encouraged to retain throughout the night. In those cases in which it is not well retained I have found it preferable to inject the oil after breakfast, following which the child is induced to lie down and retain it for at least one-half hour. He has worn an abdominal supporter and woolen underwear throughout the entire course of treatment and his mother massages his abdomen night and morning, following a rotary course beginning at the cecum.

Subsequent Course.—During the past eighteen months this boy has developed quite normally with a steadily increasing gain in weight and growth in height. At this time he is able to take a pretty general diet, limited only in the quantities of fat and sugar, his pot belly has almost entirely disappeared, his stools are normal in appearance, and he has overcome his anemia.

3. **Rickets with Secondary Dilatation of the Colon.**—Harry G., aged nine and a half years. Admitted 3/10/23.

History.—This colored boy had been artificially fed from birth, receiving condensed milk during his first five months. During the remainder of his first year he was fed various milk mixtures. At no time during his first year did he receive fresh fruit juices, vegetables, or cod-liver oil. Gives a history of having been constipated throughout his life.

Examination.—His physical examination is negative except for his marked evidences of rickets. He has a large square head, a typically rachitic chest with beading, and Harrison's groove, and greatly distended abdomen, as well as a marked deformity in all of his extremities. At the age of nine and a half years he walks with difficulty.

Fig. 90.—H. G. Rickets with secondary dilatation of the colon, showing the descending colon, sigmoid, and rectum distended by a barium enema. The megalocolon and enormous sigmoid account to a large extent for the abdominal enlargement and constipation in this case.

Roentgenologic Examination.—Shows a greatly dilated colon, sigmoid, and rectum, and late rachitic bone changes.

DIFFERENTIAL DIAGNOSIS OF THE THREE TYPES OF DILATATION OF THE COLON

In the idiopathic congenital type we have a tendency toward infantilism, emaciation with the associated enlarged abdomen.

In most cases the colon is distinctly visible through the abdominal wall and can often be palpated. Constipation with a development of fecal impaction is the most striking symptom. Diarrhea is an exceptional development. These children frequently develop acute attacks of intestinal obstruction with associated vomiting and secondary acidosis. The *second type*, celiac disease, or better termed "chronic digestive insufficiency," shows an involvement of the digestive function of the entire gastro-intestinal tract. Alternate attacks of constipation and diarrhea mark the course of this condition. Children are emaciated, show a markedly irregular weight curve, a large abdomen, and at most times copious, foul-smelling, mushy stools. At the height of the condition they have an almost complete intolerance to fat. The colon does not show the extreme degree of distention that is seen in the primary form. The *third type* of dilatation of the colon as seen secondary to rickets, while usually moderate in size, at times presents abdominal findings which may be confused with Hirschsprung's disease. The constipation in this condition which is due to an insufficiency in the musculature of the intestinal wall frequently leads to fecal impaction. These infants, however, respond very quickly to a properly constructed diet including cod-liver oil.

CLINIC OF DR. PETER BASSOE

PRESBYTERIAN HOSPITAL

EPILEPSY. WITH REPORT OF 200 CASES SEEN IN PRIVATE PRACTICE

IF we survey the present state of our knowledge of the so-called neuroses we may say that our views regarding hysteria, neurasthenia, and psychasthenia have become quite crystallized, with the main emphasis on psychic factors, whether we accept the Freudian views or not. Chorea has practically been transferred to the organic group. Epilepsy and migraine remain mysteries, and the more we learn about them and study them from every possible angle the more confused we become. In the case of epilepsy, as in that of the equally baffling dementia præcox, it begins to look as if we are not dealing with a clinical entity, but with a reaction type, a mode of response to harmful agencies of a great many varieties both somatic and psychic. To get a basis for discussion I will begin with an objective survey of some personal experience.

Statistics of 200 Cases in Private Practice Seen Between the Years of 1908 and 1918.—Nearly all of these patients were office cases. None were inmates of institutions when seen by me; 121 cases were in males; 79 in females.

The age of onset was carefully ascertained in every case, with the following result: Five years of age or under, 37; six to ten, 25; eleven to fifteen, 28; sixteen to twenty; 31; twenty-one to twenty-five, 29; twenty-six to thirty, 17; thirty-one to thirty-five, 11; thirty-six to forty, 9; forty-one to forty-five, 6; forty-six to fifty, 6; fifty-one to fifty-five, 1. It will be noticed that the onset was before the age of fifteen years in 90 cases, or 45 per cent. and before twenty years in 121 cases, or 60.5 per cent.

Hereditary Factors.—A history of epilepsy in the parents though always inquired into, was strikingly rare, and then usually only one of several possible etiologic factors. It was definite in only 4 cases.

Case I.—A man, seen at twenty-six years, had been having major attacks for one year. He had been a delicate baby, constipated all his life, and a heavy drinker for years. His mother was epileptic, his father and maternal grandfather were heavy drinkers.

Case II.—A man forty years old, suffering from diabetes mellitus, had been having a peculiar kind of epileptic attack for eight years, at intervals ranging from four to eight months. Instead of falling he would run in a dazed condition. His father had epilepsy.

Case III.—A man of twenty-five years had been having major attacks for three years, with an average interval of five weeks. About half of the attacks occurred during sleep. His father and a brother had epilepsy, and the latter committed suicide. Fourteen other brothers and sisters had died, mostly in infancy. The patient was seen in 1918. In 1923 he is reported as not having had attacks for two years, though he often drinks to excess.

Case IV.—A girl ten years old, adopted by a good family, was seen in 1919. She was backward mentally and had suffered from major convulsions since five years old. The father was an alcoholic criminal, the mother epileptic and feeble-minded, all the 4 brothers were defective. The patient still has convulsions and her mental condition is backward.

In a fifth case there was a questionable history of epilepsy in the mother; in a sixth case the father, who suffered from heart disease, had had two unconscious attacks. The following case suggests the presence of a family tendency to convulsions in infancy and early childhood.

Case V.—A female child, two years old, when seen in November, 1916 had been having occasional convulsions for a year. Her last attack occurred at the onset of mumps in 1920. She has been perfectly well ever since. Her father had convulsions in infancy, and a maternal aunt a few in her youth.

Epilepsy in other near relatives existed in 18 cases. Migraine in one or more near relatives was present in not less than 52 cases, or 26 per cent.

Alcoholism in one parent was recorded in 19 cases; insanity of some kind or other in 4 parents, and diabetes in 4. Nocturnal enuresis in a near relative existed three times.

Personal Etiologic Factors.—Difficulty at birth in some form or other had been present in only 16 cases as far as could be ascertained. The conditions encountered were: Cyanosis in a twin; cyanosis usually in a forceps case (5); breech presentation (2); premature birth with weight of 3½ pounds, large head (3); "dry" labor, forceps leaving mark on head, just "difficult," one each. The importance of these conditions becomes questionable when we find that in no case did the convulsions commence before two years after birth, the ages at onset being as follows: two years (3 cases); three years, eight years (2 cases); nine years, ten years, twelve years, thirteen years (2 cases); fourteen years, seventeen years (2 cases); twenty years, twenty-two years. Heredity and other factors were also present in many of these cases.

Head injury after birth is recorded in 12 cases. It must be remembered that this series includes only cases of ordinary epilepsy, and that I have not included cases of gross brain injury with convulsions, only part of a clinical picture of contusion of the brain.

It will be noted from Table 1 (page 522) that the other usual factors are present in about the same proportion as in the "idiopathic" cases.

The longer the interval between head trauma and onset of epilepsy, the less significance naturally can be attached to the injury.

Nocturnal Enuresis.—Apart from the seizures this was pres-

TABLE 1

TRAUMATIC EPILEPSY

Case Number.	Age when injured in years.	Interval before onset of epilepsy.	Hereditary factors.	Other factors.	Character of attacks.
1	22	4 years.	.....	.....	Minor and major.
2	9	2 days.	Mother migraine.	.....	Major nocturnal.
3	1	4 years.	.....	.....	Major nocturnal.
4	1	4 months.	.....	.....	Major and minor.
5	24, 27, 30	1 week after injury.	Brother "traumatic epilepsy."	.....	Major.
6	8	8 years.	Father alcoholic. Mother migraine.	.....	Minor and major.
7	1½	21 years.	Mother migraine.	Typhoid few months before onset.	Major nocturnal.
8	2	8 years.	.....	.....	Major recovered after craniectomy.
9	5	2 months.	.....	.....	Major nocturnal.
10	10	6 years.	.....	.....	Major.
11	13	7 months.	Mother migraine.	Cyanosis at birth.	Major.
12	12	35 years.	Father migraine (?).	.....	Major.

ent in 9 cases, just frequently enough to call attention to the long suspected relationship between enuresis, epilepsy, and migraine.

Infectious diseases immediately preceding the onset are recorded several times.

Case VI.—A boy, whose brother had had a few convulsions and recovered, had severe measles at seven years, followed by a few nocturnal convulsions, and had others at thirteen and seventeen. He has had none during the past twelve years. He was treated with bromids.

Another boy of two years, mentally deficient, developed convulsions after measles and did not improve on bromids. A third boy, four years old, whose maternal aunt and grandmother had migraine, also developed epilepsy immediately after an attack of measles.

Typhoid had preceded the onset in one patient, who also gave a history of head injury, scarlet fever in 2 cases, whooping-cough in 1 case, and 1 patient gave a history of a previous chorea. Intestinal worms were accused in 2 cases, 1 of which recovered.

Ten patients gave a history of having had one, or usually more, convulsions in infancy, with the regular epilepsy coming on later. This does not include the numerous cases with onset of the epilepsy itself in infancy or early childhood.

Endocrine Factors.—This series only permits of the recording of certain obvious endocrine features, as no routine fine scrutiny was made. By extracting every bit of evidence from these admittedly deficient records we find something suggestive of an endocrine influence for evil or good in 23 cases, as follows: Thyroid enlargement alone mentioned (5 cases); slight goiter, scanty menstruation, recovery (1); slight goiter, weight at birth 12 pounds (1); hyperthyroidism and thyroidectomy; recovery on subsequent thyroid and pituitary feeding (1); oöphorectomy at fifteen, obesity (1); uterus small, recovery after marriage (1); irregular menstruation (1); left cryptorchidism (1); polyuria (2); diabetes mellitus, normal sella (1); glycosuria after attacks (1); obesity, small sella (1); "buried" sella, many pregnancies, recovery (1); onset during pregnancy (3); aggravated by each pregnancy (1); fewer attacks during pregnancy (1).

In one woman the onset dated from puerperal eclampsia. The attacks were light and frequent, and a psychosis rapidly developed. The outcome is not known.

Mental Symptoms.—More or less deterioration of mind was recorded in 34 cases, a rather low percentage (17). As the records are especially meager on this point the figure undoubtedly should be higher. Nevertheless, the mental condition of this group is very favorable compared with any similar number of hospital or colony cases.

Character of Attacks.—The predominating type of attack may be seen from the following table (page 524). The terms "major" and "minor" are used in preference to the foreign "grand mal" and "petit mal."

TABLE 2

PREVALENT TYPES OF ATTACK

1.	Major, during waking hours	96
2.	Major, during sleep	45
3.	Minor	11
4.	Major and minor, chiefly former	10
5.	Major and minor, chiefly latter	22
6.	Psychic equivalent	1
7.	Major and psychic	3
8.	Major, minor and psychic	1
9.	Partial (Jacksonian)	7
10.	Falling only	1
11.	Running only	1
12.	Other atypical fits	2

Results.—It has been possible to follow up less than half of the cases; 23 patients are reported to have had no attacks for at least two years, and most of them have been well for over five years. As we have records of cases with recurrence after as long as six years we dare not speak of recoveries, merely of arrest of the disease. As it is of particular importance to learn the factors which make for recovery or arrest of attack we have tabulated these cases (Table 3). No particular lesson, however, can be drawn as regards any particular favorable factors except that the element of hereditary predisposition was fully as great in these cases as in the more unfavorable ones.

Quite a large number of patients report decided improvement. Many are said to have done better on luminal than on bromids.

Dislocation of the Shoulder During a Fit.—This occurred in two of the cases and was the most prominent feature in one case. Case VII, the patient a woman twenty-two years old, was sent to me by her physician on account of a supposed "neuritis" of the arms which had come on after an attack three months previously. There was plainly dislocation of both shoulders. The patient was referred to Dr. C. Hugh McKenna, who found the head of the humerus very porotic and who performed open operations on both shoulders, with good results. Incidentally, the patient has had no more convulsions.

Epilepsy as an Early Symptom of Brain Tumor.—It is impor-

TABLE 3

ARRESTED CASES

Case.	Sex.	Age at onset.	Age at last attack.	Years frec.	Hereditary factors.	Other etiologic factors.	Treatment.	Remarks.
1	M.	3	6	..	0	Worms.	Bromids.	
2	M.	2	..	8	0	Encephalitis?	Bromids.	
3	M.	7	17	12	Brother, few convulsions and recovered.	Measles before onset.	Bromids.	
4	F.	8	..	..	Mother, nervous prostration.	Cyanosis at birth.	Bromids.	
5	M.	10	11	11	0	Head trauma at two years.	Craniectomy in 1912.	Three or four light attacks after operation, then none.
6	F.	22	..	..	Parents and sister, migraine.	Small uterus.	Bromids.	Recovered after marriage.
7	M.	2	8	9	Brother, two convulsions; cousin, epilepsy; mother, maternal grandmother, and brother, migraine.	Enuresis; otitis.		
8	F.	16	..	4	Mother and two sisters, goiter.	Slight goiter. Convulsions in infancy; menstruation, scanty.	Bromids.	
9	F.	24	35	3	Migraine in several relatives.	0	Bromids, later luminal.	
10	M.	15	15	10	0	0	Bromids.	Three attacks only, 2 nocturnal.
11	M.	14	23	9	Brother, epilepsy; mother, migraine.	0	Bromids.	
12	M.	1	2	9	0	0	Bromids.	Cure credited to osteopathy.
13	M.	18	..	5	Brother, epilepsy.	0	Bromids one year.	Light attacks, head nodding. Falling attacks without convulsions.
14	F.	16	24	4	Parents, probably migraine.	Constipation.	Bromids.	
15	F.	15	34	2	Mother and maternal grandmother, migraine.	Aggravated by each pregnancy.	Therapeutic abortion.	
16	M.	17	..	..	Father and maternal aunt, migraine.	0	Bromids and adonis.	Free four years; one attack in 1920.
17	F.	21	22	7	0	0		
18	F.	1	5	3	Father, convulsions in infancy; maternal aunt, convulsions in youth.	0	Bromids.	
19	M.	22	27	2	Father and brother, epilepsy.	Alcoholism.	Luminal.	
20	F.	33	35	3	Father, alcohol.	Migraine.	Luminal.	
21	F.	39	42	5	0	Graves' disease, thyroidectomy.	Thyroid and pituitary.	
22	M.	29	48	3	Father, migraine.	Skull fracture at twelve.	Bromids, later luminal.	Attacks ceased under luminal.
23	M.	12	14	3	Maternal grandfather, alcohol.	0	Not known.	

tant to bear in mind that for a long time ordinary epileptic attacks may be the only symptom of brain tumor. The following case is noteworthy as the patient apparently recovered from his epilepsy long before tumor symptoms proper became manifest.

Case VIII.—A man, twenty years old, with negative family history was first seen in January, 1911. At sixteen years he began having minor attacks. In 1910 he began having occasional major attacks in which the head turned to the left. The only positive neurologic signs in 1911 were a right-sided ankle-clonus and a bilateral Oppenheim sign. With bromid treatment the attacks were greatly reduced in number. In June, 1912 no ankle-clonus was obtained and only a left-sided Oppenheim sign. A year or two later the attacks ceased. He was well until the summer of 1921, when he entered the University Hospital at Ann Arbor, Michigan, on account of headache, insomnia, and general weakness. Professor Carl D. Camp kindly informed me that the neurologic examination at that time was negative except for edema of the optic nerve heads without disk swelling. Roentgenologic examination of the head led to a diagnosis of a cyst in the left temporal lobe. In January, 1922 an operation was performed, and a large white mass was removed from this lobe. The central portion contained yellow fluid. The gross appearance suggested tumor, but microscopic examination of the cyst wall only revealed an inflammatory process. The patient died shortly afterward.

The Present Status of "Idiopathic" Epilepsy.—Just now our knowledge of this disorder is in the state of chaos and dissolution which, we hope, is merely preliminary to one of clarity and precision. The subject is being attacked from all sides by workers in many fields, and the result is probably going to be that there will be not one "epilepsy," but many epilepsies. We shall take up each of the principal lines of approach and try to point out the actual facts brought out.

1. **The Psychogenetic Theory.**—According to this theory, epilepsy is predetermined by the presence of the so-called

epileptic constitution, the chief psychic features of which are egocentricity, supersensitiveness, emotional poverty, and rigidity of ideation and mentation (L. Pierce Clark). Such children are wilful and prone to tantrums and are unable to meet even the ordinary difficulties of life. The fit is looked upon as a protective mechanism, the result of a desire to get away from trouble by flight into unconsciousness, or, as the psycho-analysts say, to "seek to return to the Nirvana of intra-uterine life." In fact, they claim that the epileptic cry and the cry of the newborn infant are prompted by such desire. As an instance of the protective nature of the fit they quote the famous Russian writer Dostojewsky as stating that he would give ten years of his life rather than miss the seconds of indescribable bliss preceding the lapse into unconsciousness of his own fits. We cannot accept this exclusive view, because similar mental traits exist in the victims of other psychoneuroses, such as hysteria and psychasthenia; because we consider the attacks themselves as more essential than personality traits, and because we know that causes other than psychic ones will produce convulsive seizures, in themselves indistinguishable from those of so-called true epilepsy. We do not deny that this theory may hold good for a rather small group of cases.

2. **Experimental Convulsions.**—These are usually produced by absinthe or picrotoxin, and help to throw light on the possible site of the irritative action producing the fit.

The occurrence of convulsions in decerebrated animals—from which the hemispheres and consequently all of the cortex have been removed—has been abundantly proved by Rothmann[1] and many others, both occurring spontaneously and after injection of the drugs mentioned. L. J. Pollock[2] has reviewed the literature and related new experiments. By means of pneumographic tracings he has shown that changes in respiration and blood-pressure precede aura in convulsions not only in experimental epilepsy, but in cases of the idiopathic disease. In the decerebrated animal the clonic part of the fit is usually

[1] Neurol. Centralbl. 31, 1286, 1912.
[2] Jour. Nervous and Mental Disease, 47, 114, 1918.

absent, but both Pollock and Stanley Cobb have found it present.

It is thought that in cortical Jacksonian epilepsy the occasional subsequent generalized convulsion is due to involvement of the pons and medulla (Pollock).

3. **Pathologic Anatomy and Histology.**—No uniform "pathology" of epilepsy exists, and the most experienced neuropathologist would blunder badly if asked to pick out the brains of epileptics from any miscellaneous collection. Several surgeons have made interesting observations on the exposed brains of living epileptics. Thus, Tillmann[1] states that in operations on epileptics he practically never saw a normal brain surface, and he describes increased tension of the dura, cloudiness and edema of the arachnoid—"arachnitis chronica." Similar observations have been made by Walter E. Dandy,[2] who has devised an operation consisting of peeling off the affected membrane and painting the exposed surface with a solution of iodin to prevent the recurrence of the cystic condition. So far he has made no extensive report of his results. F. Krause[3] observed the exposed brain during a fit. "Like an extremely tense bluish-red balloon it pressed against the trephine opening; there was no pulsation, no flow of fluid; the brain appeared dry." Marburg and Ranzi[4] made the same observation, with the addition that a surprisingly large amount of fluid appeared in the meninges.

A good review of our knowledge of the gross and microscopic pathology of epilepsy is given by Eugene Pollak.[5] Proliferation of the glia of the outer portion of the cortex is admitted to exist and is much stressed by Chaslin, while Alzheimer considers it secondary to destruction of cells in the second and third cell layers of the cortex. Large types of glia cells and "ameboid glia" are also seen. Developmental disorders, in the form of imperfections of brain architecture, are said to be frequent, such as displaced ganglion cells in the white matter, imperfect devel-

[1] Virchow's Arch., 229, 40, 1920.
[2] Bull. Johns Hopkins Hospital, April, 1920.
[3] Die Chir. d. Gehirns u. Ruckenmarks, 1908.
[4] Arch. f. klin. Chir., 1919, vol. 113.
[5] Jahreskurse für arztl. Fortbildung, May, 1920.

opment of the granular layers, etc. In a sense, this idea of an imperfect brain fits in with that of an "epileptic constitution" or an imperfect mental make-up.

4. **Chemical Studies in Epilepsy, Eclampsia, Spasmophilia, and Related Conditions.**—The acid-base mechanism of the blood is interesting in connection with epilepsy, as well as eclampsia and spasmophilia. R. H. Geyelin[1] found the hydrogen-ion concentration of the blood on ordinary diet more inconstant in epileptics than in normal persons, and that more convulsions in epileptics occurred when the concentration was high, *i. e.*, when the alkalinity of the blood was high. E. D. Plass,[2] in discussing eclampsia, divided the cases into hepatic and nephritic types, but found clinical distinction often to be impossible, so we probably are dealing with a common toxic factor. He looks upon eclampsia, including the convulsions, as a protective, physiologic process gone wrong, the convulsion being an expression of an effort to raise the carbon dioxid of the blood to offset the alkalosis present.

It has been shown by numerous observers that alkalosis, whether produced by excessive administration of alkalies or by forced breathing, may induce a temporary state of tetany.[3]

In a paper on changes in the acid-base equilibrium in the blood Klothilde Meier[4] states that alkalosis may develop from excessive administration of sodium bicarbonate, from regular gastric irrigations resulting in loss of hydrochloric acid, and from voluntary forced breathing (hyperpnea). While acidosis leads to coma, alkalosis leads to tetany. However, alkalosis is not always found in tetany. E. Freudenberg and P. György[5] emphasize the rôle of alkalosis in tetany and recommend the

[1] Research Assoc. in Nerv. and Ment. Dis., 1922, unpublished.

[2] Ibid.

[3] Barker, L. F., and Sprunt, T. P.: Endocrinology, January, 1922. Collip and Backus, Amer. Jour. of Physiol., April, 1920. Grant and Goldman, ibid., June, 1920. A. Goldman, Jour. Amer. Med. Assoc., April 22, 1922. K. Meier, Klin. Wchn., September 16, 1922. Freudenberg and György, ibid., February 25, 1922.

[4] Klin. Wchn., September 16, 1922.

[5] Klin. Wchn., February 25, 1922.

daily administration of 3 to 6 grams (45–90 grains) of ammonium chlorid, best given in 10 per cent. solution in divided doses at mealtime. Nine cases are related, with relief of carpopedal spasms, laryngospasms, and increased mechanical irritability.

The "dysregulatio ammoniaci" of Danish investigators (Bisgaard, Larsen), demonstrated by means of daily determination of the ammonia nitrogen and hydrogen-ion concentration in the urine, may prove of importance. It was found to be quite uniformly present in true epilepsy and tetany, not in cases of hysteria, organic brain disease, and alcoholism even when associated with convulsions. Its presence in traumatic epilepsy is considered evidence of predisposition to epilepsy.

5. **Relationship of Infantile Convulsions and Spasmophilia to Subsequent Epilepsy.**—Considering its importance, this theme is scantily treated in the literature. J. Lovett Morse[1] followed up 107 children with convulsions in infancy. He concluded that convulsions from spasmophilia are not likely to lead to epilepsy, neither are convulsions at the onset of an acute illness or acute indigestion, but those in whooping-cough and repeated minor attacks are more serious. Altogether, he finds himself unable to lay down any definite rules. D. A. Thom obtained a history of infantile convulsions in 25 per cent. of the adult epileptics at the Monson (Massachusetts) State Hospital. Of 109 cases of convulsions in children under four years, 47 have been well for at least eight years; 19 died in convulsion; 28 have convulsions, and 15 are feeble-minded. Of the 109 cases, 42 were associated with gastro-intestinal disorders and 24 of these are in the damaged group. An additional group of 29 spasmophilia cases gave 16 epileptics or feeble-minded—surprising in view of Morse's experience, all of his spasmophilia cases having remained well.

Douglas Thom[2] investigated the conditions of 431 children of 117 epileptics at the Monson State Hospital (Massachusetts) —151 of the children were dead, 19 of convulsions. Of the 280

[1] Amer. Jour. Dis. Children, August, 1919.
[2] Boston Med. and Surg. Jour., August 31, 1922.

living children, 75 per cent. of whom are over twenty years of age, 14 have had convulsions, but 6 have been free from four to twenty-four years, leaving only 8 to be considered epileptic. He concludes that "nervous and mental disorders are less frequently found in the offspring of the so-called epileptic than we have heretofore believed and the future of the offspring is not as hopeless as the pessimistic authorities on heredity have heretofore stated. Maternal defects are more frequently manifested in some form or other in the offspring than are the paternal defects, and, when present, are more likely to appear at an earlier age."

6. **The Endocrines and Epilepsy.**—Many observers have tried to attribute epilepsy to disorders of various ductless glands. Nearly thirty years ago in this country A. P. Ohlmacher called attention to the frequency of the status thymicolymphaticus in epilepsy. More recently W. Browning has reported therapeutic success from Roentgen treatment of the thymus. Bolten considered epilepsy due to the failure of the thyroid and parathyroids to detoxicate certain metabolic products. Overactivity of the adrenals has been blamed, and the hypophysis is accused by some. Thousands of ovaries have been sacrificed because the attacks in epilepsy, as in migraine, often occur at the menstrual periods. However, there is nothing very convincing about any exclusive endocrine theory.

7. **Epilepsy and Migraine as Protein Sensitization Disorders.**—This is suggested by the analogy with such periodic disorders as asthma, hay-fever, and angioneurotic edema, also by the reported relief or aggravation by pregnancy, certain foods, and the occasional beneficial effect of a powerful toxic protein, rattlesnake venom (crotalin).

Whither Are We Drifting?—As stated at the outset, the onslaught on the mysteries of dementia præcox and epilepsy from so many directions has tended to shake our belief in either being a clinical entity. Instead of speaking of the *disease* dementia præcox, we now prefer to speak merely of a schizophrenic reaction type as a response to varied psychic and physical influences. Similarly, epilepsy is on the way to be looked upon as a

reaction of a more or less protective type to a great many disturbing psychic, physical, and chemical agencies, usually only appearing in neuropathic individuals of a certain kind, possessing the "epileptic constitution."

Treatment.—If epilepsy is a "reaction type" in individuals of a certain make-up, in response to widely varied noxæ, then the therapeutic problems may be classified thus:

1. Eugenic measures to limit the number of offspring of individuals possessing the "epileptic constitution." This is very difficult, as we are not dealing so much with direct inheritance of epilepsy, as with neuropathic taint in general. Thus, it appears just as essential to forbid migraine patients to marry as those afflicted with epilepsy.

2. Measures directed against the chief precipitating factor in the given case whether this be some endocrine or metabolic imbalance or unfavorable psychic influences.

Under this heading we have the various endocrine treatments, whether by glandular products, Roentgen rays, or surgical operations. The most recent operation proposed is removal of one adrenal (Heinrich Fischer), and, as expected, this has met with little success. For "sensitization" cases we now, in addition to crotalin, have been introduced to injections of peptone. This treatment is too new to receive any more than casual mention. Calcium salts are recommended for cases on a possible spasmophilic basis.

3. Symptomatic treatment of the convulsive state: For generations bromids have been the favorite, but they are not now, having been superseded by *phenobarbital* (luminal), preferably given in a single evening dose of 1½ grains. Starvation treatment, with ingestion of plenty of water, seems to be successful in cases with a tendency to heaping of the attacks at certain periods, and the efficacy may largely be due to induction of a partial and temporary acidosis. Such surgical measures as decompression, callosal puncture, and peeling off the arachnoid of the motor cortex must also be placed in the symptomatic class.

CLINIC OF DR. ROBERT SONNENSCHEIN

POSTGRADUATE HOSPITAL

A GROUP OF NOSE AND THROAT CASES PRESENTING GENERAL SYSTEMIC SYMPTOMS

TAKING stock of one's effects and resources is advisable not only in business but in a medical way. The cases which we will show you today will be largely those which we have treated some time in the past and in which we now desire to see the ultimate result of treatment, operative or otherwise.

The subject of focal infections and their results in the body, particularly infections of the teeth, tonsils, and sinuses, has attracted the attention of the profession to a very considerable degree during the past few years. With all new movements great results are at first reported and accepted. This was the case with the Roentgen ray, radium, focal infections, vaccine therapy, and deficiency diseases which are now so much to the fore. With all these movements great enthusiasm at first prevailed and wonderful results were reported. Later on careful examinations of the histories and of the patients months and years after treatment had been given, showed that in many instances the early hopes were not realized. This has oftentimes led to complete reversal of opinion, so that many good measures have been abandoned simply because the first wave of enthusiasm subsided and discouragement followed in its wake. However, when most of these methods of treatment are carefully investigated it is found that many cases are most favorably influenced thereby, and therefore the method should be preserved and be applied to selected cases. Then the therapy receives the recognition it should have, and good results are obtained in those cases in which it should be specifically applied.

Of the foci which have received attention the tonsil surely

ranks among the most important and popular. In many cases there has been indiscriminate removal of this structure on the basis, as some have expressed it, that every tonsil is a bad tonsil. The fact that the Streptococcus viridans (as D. J. Davis has so well shown) has its natural habitat in the adenoid and tonsillar crypts has led many to assume that all tonsils are potentially foci of infection. We know, however, that as the Streptococcus viridans is almost invariably found in the tonsillar crypts, so the colon bacillus has its habitat in the intestine, and yet no one would suggest that simply because of this resections of the intestinal tract should be done. The fact of the matter is that the mere presence of the Streptococcus viridans in the tonsil or in the mouth itself does not mean that the individual will have either a local or systemic infection or disturbance.

We have several cases to present today in which the symptoms and condition of the tonsils made us feel that they were the offending factor. Tonsillectomy was performed in these cases, and we have asked the patients to return today that we may make note of their present condition as compared with the preoperative state.

Case I.—Miss I. B., aged thirty, registered nurse, was referred to this clinic by an ophthalmologist with the history that she had intermittent paresis of the left ciliary muscle. Careful examination of the eyes had been made before the ophthalmologist referred her to us in the belief that the condition was due to focal infection. Two years ago the patient suffered from a streptococcic sore throat, but has had no further trouble since. There was no history of nasal infection.

Examination of the nose showed some intumescences of the inferior turbinates and large middle turbinates. Transillumination of the sinuses was negative. The nasopharynx was negative. The tonsils were flat and submerged and the crypts contained a great deal of caseous purulent material. In the neck a number of enlarged glands could be palpated. On the basis of an absence of findings by the internist in other parts of the body, together with the history of a streptococcus sore throat,

and the presence in the crypts of caseous purulent secretion, we removed the tonsils under local anesthesia with knife and snare one month ago. The anesthesia was that which we usually employ in local cases. Mention of it here will save the repetition in the other cases to be presented. The posterior wall of the pharynx and the anterior pillars of the fauces and the hard palate were swabbed twice with a small amount of 10 per cent. cocain solution in order to lessen the pharyngeal reflexes and also to make the insertion of the needle for infiltration anesthesia less painful. In less apprehensive persons cocain is entirely omitted. Then a 1 per cent. solution of apothesine (4 drams—16 c.c.) to which 10 to 12 drops of a 1 : 1000 solution of adrenalin had been added was injected with a straight needle into the region of the posterior palatine nerve just internal to the last upper molar tooth (about 7 drops—0.5 c.c.) and the remainder into the areolar tissue just external of the capsule of the tonsil. Most of the solution was injected at the junction of the upper and middle thirds of the space and some of it at the base of the tonsil. Then with the knife and snare the tonsils were removed.

Today the patient is before us and the headaches and symptoms of the eye condition previously complained of have wholly disappeared. The ophthalmologist also states in his letter that he finds the eyes entirely normal.

Case II.—Mrs. J. W. H., aged thirty-four years, housewife, was referred to us from the Medical Department with a history of having pains in the joints, chiefly in the shoulders and knees. About once a year the patient has been subjected to a severe attack of such trouble. *x*-Ray examination of the teeth has been negative. There has been some nasal obstruction, but no definite history of suppuration. Examination of the urine is entirely negative.

Examination revealed a deflection of the septum to the left with some intumescence of the inferior turbinates. Transillumination of the sinuses was negative. The tonsils were found deeply submerged and containing caseous and purulent material in the crypts.

daily administration of 3 to 6 grams (45–90 grains) of ammonium chlorid, best given in 10 per cent. solution in divided doses at mealtime. Nine cases are related, with relief of carpopedal spasms, laryngospasms, and increased mechanical irritability.

The "dysregulatio ammoniaci" of Danish investigators (Bisgaard, Larsen), demonstrated by means of daily determination of the ammonia nitrogen and hydrogen-ion concentration in the urine, may prove of importance. It was found to be quite uniformly present in true epilepsy and tetany, not in cases of hysteria, organic brain disease, and alcoholism even when associated with convulsions. Its presence in traumatic epilepsy is considered evidence of predisposition to epilepsy.

5. **Relationship of Infantile Convulsions and Spasmophilia to Subsequent Epilepsy.**—Considering its importance, this theme is scantily treated in the literature. J. Lovett Morse[1] followed up 107 children with convulsions in infancy. He concluded that convulsions from spasmophilia are not likely to lead to epilepsy, neither are convulsions at the onset of an acute illness or acute indigestion, but those in whooping-cough and repeated minor attacks are more serious. Altogether, he finds himself unable to lay down any definite rules. D. A. Thom obtained a history of infantile convulsions in 25 per cent. of the adult epileptics at the Monson (Massachusetts) State Hospital. Of 109 cases of convulsions in children under four years, 47 have been well for at least eight years; 19 died in convulsion; 28 have convulsions, and 15 are feeble-minded. Of the 109 cases, 42 were associated with gastro-intestinal disorders and 24 of these are in the damaged group. An additional group of 29 spasmophilia cases gave 16 epileptics or feeble-minded—surprising in view of Morse's experience, all of his spasmophilia cases having remained well.

Douglas Thom[2] investigated the conditions of 431 children of 117 epileptics at the Monson State Hospital (Massachusetts) —151 of the children were dead, 19 of convulsions. Of the 280

[1] Amer. Jour. Dis. Children, August, 1919.
[2] Boston Med. and Surg. Jour., August 31, 1922.

living children, 75 per cent. of whom are over twenty years of age, 14 have had convulsions, but 6 have been free from four to twenty-four years, leaving only 8 to be considered epileptic. He concludes that "nervous and mental disorders are less frequently found in the offspring of the so-called epileptic than we have heretofore believed and the future of the offspring is not as hopeless as the pessimistic authorities on heredity have heretofore stated. Maternal defects are more frequently manifested in some form or other in the offspring than are the paternal defects, and, when present, are more likely to appear at an earlier age."

6. **The Endocrines and Epilepsy.**—Many observers have tried to attribute epilepsy to disorders of various ductless glands. Nearly thirty years ago in this country A. P. Ohlmacher called attention to the frequency of the status thymicolymphaticus in epilepsy. More recently W. Browning has reported therapeutic success from Roentgen treatment of the thymus. Bolten considered epilepsy due to the failure of the thyroid and parathyroids to detoxicate certain metabolic products. Overactivity of the adrenals has been blamed, and the hypophysis is accused by some. Thousands of ovaries have been sacrificed because the attacks in epilepsy, as in migraine, often occur at the menstrual periods. However, there is nothing very convincing about any exclusive endocrine theory.

7. **Epilepsy and Migraine as Protein Sensitization Disorders.**—This is suggested by the analogy with such periodic disorders as asthma, hay-fever, and angioneurotic edema, also by the reported relief or aggravation by pregnancy, certain foods, and the occasional beneficial effect of a powerful toxic protein, rattlesnake venom (crotalin).

Whither Are We Drifting?—As stated at the outset, the onslaught on the mysteries of dementia præcox and epilepsy from so many directions has tended to shake our belief in either being a clinical entity. Instead of speaking of the *disease* dementia præcox, we now prefer to speak merely of a schizophrenic reaction type as a response to varied psychic and physical influences. Similarly, epilepsy is on the way to be looked upon as a

reaction of a more or less protective type to a great many disturbing psychic, physical, and chemical agencies, usually only appearing in neuropathic individuals of a certain kind, possessing the "epileptic constitution."

Treatment.—If epilepsy is a "reaction type" in individuals of a certain make-up, in response to widely varied noxæ, then the therapeutic problems may be classified thus:

1. Eugenic measures to limit the number of offspring of individuals possessing the "epileptic constitution." This is very difficult, as we are not dealing so much with direct inheritance of epilepsy, as with neuropathic taint in general. Thus, it appears just as essential to forbid migraine patients to marry as those afflicted with epilepsy.

2. Measures directed against the chief precipitating factor in the given case whether this be some endocrine or metabolic imbalance or unfavorable psychic influences.

Under this heading we have the various endocrine treatments, whether by glandular products, Roentgen rays, or surgical operations. The most recent operation proposed is removal of one adrenal (Heinrich Fischer), and, as expected, this has met with little success. For "sensitization" cases we now, in addition to crotalin, have been introduced to injections of peptone. This treatment is too new to receive any more than casual mention. Calcium salts are recommended for cases on a possible spasmophilic basis.

3. Symptomatic treatment of the convulsive state: For generations bromids have been the favorite, but they are not now, having been superseded by *phenobarbital* (luminal), preferably given in a single evening dose of 1½ grains. Starvation treatment, with ingestion of plenty of water, seems to be successful in cases with a tendency to heaping of the attacks at certain periods, and the efficacy may largely be due to induction of a partial and temporary acidosis. Such surgical measures as decompression, callosal puncture, and peeling off the arachnoid of the motor cortex must also be placed in the symptomatic class.

CLINIC OF DR. ROBERT SONNENSCHEIN

POSTGRADUATE HOSPITAL

A GROUP OF NOSE AND THROAT CASES PRESENTING GENERAL SYSTEMIC SYMPTOMS

TAKING stock of one's effects and resources is advisable not only in business but in a medical way. The cases which we will show you today will be largely those which we have treated some time in the past and in which we now desire to see the ultimate result of treatment, operative or otherwise.

The subject of focal infections and their results in the body, particularly infections of the teeth, tonsils, and sinuses, has attracted the attention of the profession to a very considerable degree during the past few years. With all new movements great results are at first reported and accepted. This was the case with the Roentgen ray, radium, focal infections, vaccine therapy, and deficiency diseases which are now so much to the fore. With all these movements great enthusiasm at first prevailed and wonderful results were reported. Later on careful examinations of the histories and of the patients months and years after treatment had been given, showed that in many instances the early hopes were not realized. This has oftentimes led to complete reversal of opinion, so that many good measures have been abandoned simply because the first wave of enthusiasm subsided and discouragement followed in its wake. However, when most of these methods of treatment are carefully investigated it is found that many cases are most favorably influenced thereby, and therefore the method should be preserved and be applied to selected cases. Then the therapy receives the recognition it should have, and good results are obtained in those cases in which it should be specifically applied.

Of the foci which have received attention the tonsil surely

ranks among the most important and popular. In many cases there has been indiscriminate removal of this structure on the basis, as some have expressed it, that every tonsil is a bad tonsil. The fact that the Streptococcus viridans (as D. J. Davis has so well shown) has its natural habitat in the adenoid and tonsillar crypts has led many to assume that all tonsils are potentially foci of infection. We know, however, that as the Streptococcus viridans is almost invariably found in the tonsillar crypts, so the colon bacillus has its habitat in the intestine, and yet no one would suggest that simply because of this resections of the intestinal tract should be done. The fact of the matter is that the mere presence of the Streptococcus viridans in the tonsil or in the mouth itself does not mean that the individual will have either a local or systemic infection or disturbance.

We have several cases to present today in which the symptoms and condition of the tonsils made us feel that they were the offending factor. Tonsillectomy was performed in these cases, and we have asked the patients to return today that we may make note of their present condition as compared with the preoperative state.

Case I.—Miss I. B., aged thirty, registered nurse, was referred to this clinic by an ophthalmologist with the history that she had intermittent paresis of the left ciliary muscle. Careful examination of the eyes had been made before the ophthalmologist referred her to us in the belief that the condition was due to focal infection. Two years ago the patient suffered from a streptococcic sore throat, but has had no further trouble since. There was no history of nasal infection.

Examination of the nose showed some intumescences of the inferior turbinates and large middle turbinates. Transillumination of the sinuses was negative. The nasopharynx was negative. The tonsils were flat and submerged and the crypts contained a great deal of caseous purulent material. In the neck a number of enlarged glands could be palpated. On the basis of an absence of findings by the internist in other parts of the body, together with the history of a streptococcus sore throat,

and the presence in the crypts of caseous purulent secretion, we removed the tonsils under local anesthesia with knife and snare one month ago. The anesthesia was that which we usually employ in local cases. Mention of it here will save the repetition in the other cases to be presented. The posterior wall of the pharynx and the anterior pillars of the fauces and the hard palate were swabbed twice with a small amount of 10 per cent. cocain solution in order to lessen the pharyngeal reflexes and also to make the insertion of the needle for infiltration anesthesia less painful. In less apprehensive persons cocain is entirely omitted. Then a 1 per cent. solution of apothesine (4 drams—16 c.c.) to which 10 to 12 drops of a 1 : 1000 solution of adrenalin had been added was injected with a straight needle into the region of the posterior palatine nerve just internal to the last upper molar tooth (about 7 drops—0.5 c.c.) and the remainder into the areolar tissue just external of the capsule of the tonsil. Most of the solution was injected at the junction of the upper and middle thirds of the space and some of it at the base of the tonsil. Then with the knife and snare the tonsils were removed.

Today the patient is before us and the headaches and symptoms of the eye condition previously complained of have wholly disappeared. The ophthalmologist also states in his letter that he finds the eyes entirely normal.

Case II.—Mrs. J. W. H., aged thirty-four years, housewife, was referred to us from the Medical Department with a history of having pains in the joints, chiefly in the shoulders and knees. About once a year the patient has been subjected to a severe attack of such trouble. *x*-Ray examination of the teeth has been negative. There has been some nasal obstruction, but no definite history of suppuration. Examination of the urine is entirely negative.

Examination revealed a deflection of the septum to the left with some intumescence of the inferior turbinates. Transillumination of the sinuses was negative. The tonsils were found deeply submerged and containing caseous and purulent material in the crypts.

In this case tonsillectomy was done under general anesthesia, as the patient was exceedingly apprehensive and violently opposed to local anesthesia. As you know from experience in this clinic we are very much in favor of local anesthesia in tonsillectomy wherever it can be used, and as a rule employ it in most persons aged fourteen or fifteen years upward. Only in those instances where the patient is too apprehensive, or where the pharyngeal reflex is too marked, so that we fear it will be impossible to control the patient during operation, do we resort to a general anesthetic.

This patient made an uneventful recovery and today we learn that the pains in the joints previously complained of have disappeared. Needless to say, no medicine in the way of sedatives or alteratives was used which might in any way have influenced these pains. The only thing that was done was the tonsillectomy.

Case III.—Mrs. M. H. E., aged forty-eight, housewife, told us on her first appearance in the clinic that one month previously she experienced the first attack of sore throat which she had had in many years. Her chief trouble lay in backache and occasionally pains in the joints. *x*-Ray examination showed a beginning lipping of the lower dorsal vertebræ.

Examination of the nose was negative except for some intumescence of the inferior turbinates. Transillumination of the sinuses was likewise negative. The tonsils were submerged and contained some caseous material. The neck showed a few palpable lymph-glands.

In this case we apparently had to deal with an arthritis, possibly a beginning arthritis deformans, but at any rate a change in the intervertebral joints which had produced the previously mentioned lipping. Owing to the fact that no other focus of infection could be found, the internist was very desirous that the tonsils be removed, and this was done.

We now find that the pains have entirely disappeared. Of course the changes in the vertebræ have not been influenced by the tonsillectomy, but will, we trust, remain in the condition in

which they are today, instead of becoming worse, as would probably have happened without the operative intervention.

Case IV.—Mrs. F. K. S., aged thirty-five, housewife, gave us the history that for almost a year she had suffered from intermittent pains confined, strange as it may seem, entirely to the right side of the body: in the right shoulder, the upper arm, in the right side of the back, right hip and thigh. On palpation of the parts there was no tenderness to the touch. An aching and at times boring pain often appeared, lasting anywhere from a few minutes to many hours. These attacks would at times recur every day for a week or two, and then might be absent for several weeks.

This patient was most carefully examined from head to foot by various specialists. The sinuses were transilluminated and *x*-rayed and found negative. The teeth were also *x*-rayed and found entirely free from apical and other abscesses. A complete examination by an internist and another by a gynecologist were made, but no definite source of the trouble could be found. The patient stated that she had a rather severe attack of tonsillitis two years ago, and that this was the first one she had experienced.

Examination of the nose at the time she entered the clinic was entirely negative. The tonsils were small and almost completely submerged, so that it was impossible to see the crypts until the tonsil was everted by pressure against the anterior pillar. A small amount of secretion could then be expressed from the crypts. In view of the negative findings previously mentioned we decided that the tonsils must be the offending factors. They were removed under local anesthesia and within one month's time the patient was entirely free from trouble without any further treatment.

These 4 cases are most happy ones in that by exclusion in all instances, and in one or two instances by the finding of caseous purulent material in the crypts, the indication for tonsillectomy was made. In these cases most satisfactory results were obtained. It must be remembered, however, that the mere pres-

ence of caseous material in the crypts does not mean that the tonsil is diseased. These caseous masses consist largely, as you know, of mixtures of desquamated epithelial cells from the mucosa, particles of food, bacteria and débris, and are found in a great many tonsils which never give the individual any trouble. When, however, this material is not merely caseous, but has a purulent character, and when we have together with it persistent redness of the anterior pillars and at times enlarged cervical lymph-glands; and if, furthermore, there is a history of either previous attacks of tonsillitis or complications and symptoms which by exclusion cannot be assigned to infection in any other organ in the body, then we believe that complete tonsillectomy properly performed will in many instances afford great relief from the annoying symptoms.

A very important point to be remembered in therapeutics is that many individuals show idiosyncrasies toward one or another drug. Just as we know that certain foods will produce cutaneous, intestinal, and other reactions (for instance, fresh corn, strawberries, lobster, and other sea food, etc.) in one individual, while they can be tolerated to almost any extent by others, so we know that with drugs the same dictum applies. Many individuals cannot take even the slightest amount of belladonna or its preparations without great dryness of the throat, flushing of the skin, or mental disturbances. Some persons are unable to take even the smallest fraction of a grain of quinin without having tinnitus aurium, etc. In these days vast quantities of the salicylates are being used by patients with or without the advice of the physician. One preparation in particular has in the last fifteen or twenty years become exceedingly popular with the laity, namely, acetosalicylic acid (the acetic acid ester of salicylic acid), commonly known under its trade name of aspirin. This is being consumed in almost incredible amounts. While it is well known that the salicylates can produce and often do cause temporary tinnitus aurium, it seldom happens, we believe, that patients seek the advice of a clinic or physician merely because of this one symptom unless it is due, as it most often is, to an ear condition itself. Often the cause

is a change in the auditory nerve, a middle-ear affection, or a combination of these. In most instances where salicylates are being used they are being employed only for a short time. The tinnitus is then likewise transitory, but we have a patient to present today in whom noises in the ear have been of long standing.

Case V.—Miss M. G., aged forty-nine, came to the clinic some weeks ago with a history of having had marked tinnitus in both ears, especially the left one, for the past year or more, and there has also been some diminution in hearing. She tells us that for twelve years or more she has been taking aspirin quite frequently because of severe headaches (usually frontal in character). These pains are apparently not associated with any nasal condition, but usually appear at the time of menstruation, beginning some time before, and lasting during and continuing for some time after this period. Rarely does the patient have two weeks' time of freedom from headaches. These pains are often accompanied by some nausea and are usually relieved by sleep. Two years ago the tonsils were removed.

Examination of the nose showed a moderate deviation of the septum. The pharynx was entirely free from evidence. Transillumination of the sinuses was negative. The drum membranes showed slight cloudiness. Complete functional testing of the ears was carried out. The whispered voice was heard a distance of 5 meters in each ear. The range of hearing for the tuning-forks was from lowest C_1 to highest C^5. The Rinné was positive and bone conduction was normal. In a word, we did not find here symptoms of degeneration of the auditory nerve which are so often responsible for the production of tinnitus aurium. It is true that a diseased middle ear or eustachian tube often produce tinnitus, but as a rule this is of low pitch and is usually aggravated by nasal catarrh, and infections in the nasopharynx or in the middle ear itself. Of course, in cases of otosclerosis a very persistent and distressing tinnitus is present which is not influenced by medication or otherwise. In this instance, however, as we have shown by the functional testing

of the ears, neither infection of the middle ear nor of the internal ear itself could be demonstrated. We assumed that the tinnitus was due to the long-continued use of aspirin and so advised the patient. She was told entirely to discontinue it and to use in its place pyramidon (aminopyrin).

Now after several months the patient tells us, as you have heard, that the tinnitus is entirely gone; the pyramidon has relieved the headache, but has produced no noises in the ear. Therefore in cases of tinnitus aurium where no quinin has been used, and where the functional testing of the ears shows neither middle nor inner ear disease, be sure to learn whether one or the other of the salicylates has been taken for a considerable period of time.

Case VI.—Angioneurotic edema is a peculiar condition in which without any apparent cause sudden swelling may occur in any part of the body. Very often the sites involved will be different with each attack. For instance, at one time the left cheek of the individual may suddenly swell, and the next time the right leg, etc. Where this edema occurs in the upper respiratory tract, particularly in the larynx or trachea, the results are often most dire, and if not promptly recognized and relieved death from asphyxiation may occur.

Miss J. S., aged nineteen, student, appeared before us about six months ago with a history of having had the previous night a sudden swelling of the right cheek and the tissues about the right eye. This was the third attack she had had in three months, and the only difference was that in the other seizures the swelling completely closed the eye, whereas this time it was not quite so extensive. There were at that time no apparent inflammatory signs or symptoms in the eye, cheek, or neck. The skin of the cheek itched a great deal. The patient stated that she was not particularly susceptible to foods such as fish, lobsters, cheese, etc. During the past few years she has had each winter an attack of cervical adenitis, but rarely has sore throat or any head cold.

Our records show that the examination at that time revealed

a marked edema below the right eye and over the right cheek. The nose showed some intumescence of the inferior turbinates, but both transillumination and inspection of the sinuses were negative. The tonsils were found to be small and submerged, but contained no pus. The patient was at that time instructed to use ice applications, and tablets containing 1/600 grain of atropin were given with instructions to take two of them every two hours until the swelling diminished or the throat became somewhat dry. She reported to us that this therapy very promptly relieved the symptoms. At that time we considered the advisability of having sensitization tests made in order to see whether this was a case of protein sensitization. The patient, however, did not return, and so we were unable to carry out this measure.

On questioning her today we learn a fact which she failed to reveal before, namely, that each attack of this edema had followed the use of aspirin. This also brings out an important practical point, namely, that it is very difficult unless special effort is made to obtain all the essential facts in a patient's history which have a direct bearing on the case. Patients, like witnesses, oftentimes tell the truth, but not the whole truth. Not infrequently they fail to mention facts which though apparently trivial may actually be the determining factor so far as the diagnosis is concerned. On the other hand, it is of course remiss in the doctor and was perhaps in our case that we did not diligently enough inquire regarding the use of drugs aside from informing ourselves about the patient's toleration of these various foods.

There is still a considerable tendency in medicine to treat the prominent symptoms instead of the cause thereof. Many instances of cough are treated on the basis of bronchial origin. We have seen instances in this clinic where patients had been taking sedatives for weeks and weeks, due largely to the fact that careful examination of the chest had not been made and bronchitis eliminated, or where examination was carried out and no bronchitis found, but still the sedatives were given. It has been our experience that many cases of cough have been due not

primarily to a pulmonary condition, but that infection higher up has been the source of the trouble and has produced the bronchial irritation. We have a few cases to show you today in which there has been severe and rather persistent cough due primarily to a nasal and not pulmonary infection.

Case VII.—Dr. J. S., aged twenty-five, dentist, came to us a month ago with the history of having had a severe bronchial cough for several months. He also had a history of a nasal obstruction and some impairment of hearing for some years past. Examination of the nose revealed a rather marked deviation of the septum to the left with large inferior turbinates. The nasopharynx was negative at this examination, but a few days later showed mucopus. Transillumination of the sinuses revealed cloudy maxillary antra, especially the left one. The tonsils were flat and submerged, but contained no pus. In view of the fact that the transillumination finding was so definite it was not considered necessary to have an *x*-ray taken of the sinuses. Both antra were irrigated by way of the natural opening in the middle meati and much pus evacuated from each one. After four or five irrigations at intervals of two to three days the cough entirely disappeared without the use of any other therapeutic measure, and the patient has remained free from symptoms since this time.

Case VIII.—Miss I. G., aged twenty-two, stenographer. Several years ago this patient had a marked hypertrophic rhinitis for which conservative surgery on the inferior turbinate had been performed. At the same time there was a chronic ethmoiditis, for the relief of which the anterior ethmoid cells had been opened. A few months ago she reappeared after an interval of seven years with entire freedom from symptoms and with the history that a few weeks before returning she had developed a very severe cough together with nasal obstruction and headaches. The chest had been examined by an excellent internist and reported negative. Transillumination of the sinuses showed both maxillary antra dark. The tonsils were red and there was

some pus in the crypts. There was also considerable injection of the mucosa covering the epiglottis. *x*-Ray of the teeth merely showed an unerupted second upper right bicuspid, but no apical or other disease. The patient's condition was deplorable in that she had a most distressing cough which practically prevented sleeping. She also had a slight amount of fever, ranging from 99.4° to 100° F. per mouth. Irrigation of the maxillary sinuses by way of the natural opening in the middle meatus revealed an enormous quantity of pus. Owing to the acute inflammatory condition in the pharynx, larynx, and trachea topical applications were made to these parts. Within a week of the beginning of irrigation the cough markedly diminished and within two weeks was entirely gone. There can be no question that the flowing down from the nose of great quantities of pus infected the pharynx, larynx and trachea, and the bronchial mucosa.

Case IX.—The third case of this series is quite similar, but somewhat less severe. Mr. M. F., aged thirty-nine, a merchant, came to us some weeks ago with the history of having a rather distressing dry cough, particularly at night. The nose showed a marked S-shaped deviation of the septum to both sides, with considerable mucopus in both nares. The nasopharynx likewise contained mucopus. The uvula was quite long and thick. The tonsils were absent, having been removed some years ago. The patient gave a history of having had six years previously a retropharyngeal abscess which had descended and had markedly interfered with the larynx. This was opened after several days' time, with complete relief. Shortly thereafter the patient had had his tonsils removed, and since then he had had no further trouble until the one which we have just recited.

Transillumination showed a shadow over the left maxillary sinus. Irrigation of the cavity by way of the middle meatus revealed considerable mucopus. After a half-dozen treatments at intervals of two days the cough disappeared and he is now entirely free from any distress.

These rather simple examples should serve to emphasize the

fact that a careful examination of the nose, its accessory sinuses, and of the nasopharynx should be carried out in all cases in which cough or other symptoms referable to the lower portion of the respiratory tract are present. Of course not all instances of cough are caused or influenced by nasal suppurative conditions. The majority of coughs probably are due to some laryngeal, tracheal, or bronchial infection. Cardiac conditions by producing passive congestion may also cause a cough, but we should never forget that an acute infective process in the upper portion of the respiratory tract may to a large extent be responsible for the condition. A further point, the mere treatment by means of sedatives which influence the bronchial mucosa will in no wise lessen the disease, but simply serve probably to derange the patient's gastro-intestinal tract without in any way affecting the nasal process.

Case X.—The last case which we wish to show you today is most interesting in that severe headaches persisted for a long time before the etiologic factor could be determined.

Miss M. G., aged eighteen, stenographer, was referred to this clinic by a most excellent internist, with the request that we solve the problem of the severe headaches which had persisted for over a year. These pains are most often over both frontal regions, at times over the vertex, and occasionally radiate to the shoulder. Careful examination of the eyes with refraction, etc., had failed to show any trouble in these structures. There was no history of nasal obstruction or discharge. The tonsils had been removed two years ago because of frequent sore throats. The headaches usually began on awakening and became worse toward evening. The internist reported that a complete examination of the body gave only negative findings.

Examination of the nose showed some intumescences of the inferior turbinates. The middle turbinates lay very close to the lateral nasal wall. There was some tenderness externally over the right supra-orbital notch. Transillumination of the sinuses was negative. The nasopharynx and pharynx were negative. The tonsils, as aforestated, had been entirely removed. The

lingual tonsil was rather thick, but showed no other changes. The pharyngeal reflex was considerably diminished. The larynx and trachea were negative. There was some moderate enlargement of the thyroid gland. The Wassermann reaction was reported negative. Thus far we had found absolutely no cause for the headache. Examination of the mouth showed that both lower third molars were unerupted. *x*-Ray picture proved these two teeth markedly impacted. There was also an abscess of the upper left second bicuspid.

Under novocain anesthesia both of the lower third impacted molars were removed by an extracting dentist, and the patient now has returned to us with the history that only occasionally does she have a slight headache. The severe almost daily headaches have disappeared. Previous to removal of the teeth all sorts of sedatives had been used without avail. It must be remembered that very often headaches are reflex in character. The actual cause may be in the teeth or elsewhere without any disease of the nasal accessory sinuses, to which focus we as rhinologists are so much in the habit of referring most of the headaches which we see. Headache, like any other pain, is merely a symptom either of local or systemic origin. Disease remote from the head may by the entrance of deleterious substances from that part into the blood-stream produce headache. Cardiac conditions with passive congestion, nephritis, diabetes, gastro-intestinal disturbances, etc., will oftentimes produce headache when locally we can find absolutely no evidence within the cranium. Do not forget to make a careful inspection of the teeth or to have the same done by a competent dentist, together with an *x*-ray picture of the jaw; for often when the teeth so far as their crowns are concerned seem perfectly normal, diseased conditions either at the apices or the presence of an unerupted tooth or teeth, such as we found in this last case, will be wholly or at least partly responsible for the headache. Treat the condition, wherever you can find it, rather than the symptom.

CLINIC OF DR. NATHAN SMITH DAVIS, III

MEDICAL DISPENSARY CLINIC, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

ARTERIAL HYPERTENSION: ITS RELATIONSHIP TO CHRONIC INTESTINAL DISORDERS

Case I.—R. H., 27,268D, female, negro, widow, aged twenty-eight, was admitted to the Northwestern Dispensary March 16, 1923, complaining of, first, shortness of breath on exertion for the past four weeks, beginning when she had a cold with fever, pain in joints, and cough; second, pain in the epigastrium referred to the right and left flanks, coming on from a few minutes to several hours after meals, and present for about two weeks; third, slight sore throat.

For the last month or so the patient's appetite has been poor. The bowels move regularly without laxatives, but are mushy and have been so most of the time for the past year.

Previous Illnesses.—She had influenza in 1912, measles in childhood, and gonorrhea in 1919.

Menstrual History.—Menses are regular, accompanied by slight vertigo, profuse, and last from four to five days.

Family History.—Mother is living, aged seventy-three, has arterial hypertension, with chronic constipation; father died of dropsy; she has two brothers living, one is nervous; the other has stomach trouble; five sisters, one is nervous; one operated on recently and uterine fibroid removed; three well; two sisters and one brother dead, cause not known; husband divorced, was well when last heard from; no pregnancies.

The patient is a rather obese young colored woman who does not appear ill. Eyes react to light and accommodation; ears negative; mouth, teeth in bad condition, pyorrhea, and

caries; tonsillitis, chronic hypertrophic. Cervical lymph-glands and thyroid not enlarged on palpation. Chest symmetric, expansion equal, no dulness, no râles or altered breath sounds, except slightly prolonged expiration over larger bronchi. Heart, apex-beat in sixth interspace 12 cm. to left of midsternal line on percussion. Systolic murmur heard over precordium, aortic second greater than pulmonary second sound.

The systolic blood-pressure was 188, diastolic 128; pulse 72 regular; temperature 98.2° F.

The abdomen was flaccid, no tenderness, no mass; liver and spleen not palpable; reflexes normal.

The specimen of urine was cloudy, straw colored, alkaline, contained trace of albumin and many pus-cells, and squamous epithelial cells. No casts were found. Specific gravity was 1017.

Treatment.—The patient was given low protein, low salt diet, with an abundance of fruit and vegetables, especially greens, and thyroid extract, 2 grains, after meals for two weeks, and lactose, 8 gm., after meals for two weeks.

On March 16th blood-pressure was 188/128; on the 23d, 158/118; on the 30th, 148/110.

Case II.—C. R., female, white, single, aged forty-one in 1896. The patient consulted Dr. N. S. Davis, Jr., in 1896 for hydrops of the knee. In November, 1902 for abrasion of the hand. In June, 1903 she returned, complaining of belching, nervousness, and constipation, for which she received dietary and medicinal treatment during the rest of that year.

On December 18, 1920 she consulted me on account of an infected hand. At this time she also complained about getting up several times at night to urinate and was somewhat troubled with constipation.

On examination, there was an infected birthmark on the left hand, with some axillary lymphadenopathy. The mouth was negative; the lungs were negative; the heart was not appreciably enlarged, the heart tones were clear, with the aortic second accentuated. The abdomen was flaccid and there was some tenderness over the descending colon and sigmoid, which

were palpable. The pulse was 72, regular, and of good quality. The systolic blood-pressure was 200, the diastolic 110.

On December 27, 1920 she returned, with conditions all improved. The systolic blood-pressure was 180, diastolic blood-pressure 110; pulse 68, after ten days on low protein salt-free diet. Urine at this examination had specific gravity of 1012 and contained no albumin, casts, or sugar.

Case III—M. H. G., female, white, single, aged twenty-five in 1892. The patient consulted Dr. N. S. Davis, Jr., on January 12, 1892, complaining of a coryza and constipation. In August, 1892 for some small ulcerations on the right arm and body which started as small furuncles. In April, 1893 for indigestion, heartburn, and constipation. She was married between this time and April, 1900, when she first had a bronchitis. In 1909 again for a bronchitis and constipation, for both of which she was under treatment off and on until 1918.

On January 7, 1921 she consulted me on account of a cold of the type usually followed by bronchitis. At this time she also complained of mushy stools; borborigmus; numbness of the arms and fingers, especially at night; swelling of the feet at night; nocturia and dyspnea on exertion; also stated that she had been gaining weight for the last two years.

On examination, the patient was a very obese large woman, aged fifty-eight, with quite a marked growth of hair on the face. There was a nasopharyngitis and acute bronchitis. The heart was not enlarged on percussion. There were no murmurs and the aortic second sound was markedly accentuated. The abdomen was distended, but there was no rigidity and only slight tenderness over the colon. Systolic blood-pressure was 190, diastolic blood-pressure 110; pulse 88.

She was put on meat-free, low salt diet. On January 21, 1921 she was feeling much better, with a blood-pressure of 165/95 and a pulse of 92. On March 10, 1921 she had another attack of acute pharyngitis and bronchitis. At this time her blood-pressure was 162/104 and pulse 98. On November 12, 1921 she had a bronchitis and pharyngitis. Her blood-pressure

had dropped to 135/80, with a pulse-rate of 76. On January 26, 1922 she had another cold and a little constipation. There was marked rise in the blood-pressure to 180/100, with a pulse of 96. February 21st she had a subacute pharyngitis. Blood-pressure was 140/80. She was seen again on August 28th, at which time she was suffering from a cold; blood-pressure was 150/90. On December 27th she had an acute bronchitis and constipation for a few days, with a blood-pressure of 160/80.

Case IV.—A. N., white, female, married, aged fifty-eight. Since the spring of 1922 she has had urticarial eruptions from time to time. She feels tired, nervous, and complains of rheumatic pains in the extremities, with some weakness. She has had no sore throat or trouble with her teeth. She complains of belching after meals and chronic constipation of many years' standing. Formerly she had vertigo at times. She had the menopause eleven years ago.

Physical examination showed the lungs negative and the heart tones clear, with the aortic second greater than the pulmonary, but not accentuated.

She was put on a low protein, low salt diet, and given petrolatum liquidum internally. Blood-pressure on September 21, 1922 was 200/110; pulse 68 and regular. By September 26th blood-pressure had dropped to 165/100, with a pulse of 68. At this time she was suffering from an ulcerated tooth. On October 12th blood-pressure was 170/90, pulse 76 and regular, and she was feeling better in every way. When seen again on November 10, 1922 she complained of some vertigo and urticaria. Blood-pressure was 168/90. Pulse was 70 and there were a few ventricular premature systoles. She was still constipated. On December 18, 1922 there was some periarthritis of fingers, urticaria, and a little vertigo. Bowels were more regular. Blood-pressure was 150/85 and pulse 68 and regular. Urinalysis showed a specific gravity of 1021, no albumin or sugar, highly acid, no casts.

It will be noted that in the above patients with high blood-pressures there is a history of long-standing constipation or other

intestinal trouble. Gastro-intestinal symptoms are so common in patients with arterial hypertension that a study of 55 successive cases seen in private practice was made to ascertain its frequency; 35 of the patients in this group were women and 20 men. About two-thirds of them were overweight and in about one-third there was marked obesity. Two of the patients were under forty; 10 were between forty-one and fifty; 19 between fifty-one and sixty; 17 between sixty-one and seventy, and 3 were over seventy. In 4 cases the ages were not recorded.

Fifty of the patients in this series of 55 had a history of some chronic gastro-intestinal trouble, such as constipation, enterocolitis, cathartic colitis, for from a few months to thirty years before the hypertension was discovered. In 5 the record made no mention of any gastro-intestinal symptons. In many of the cases access was had to the records of Dr. N. S. Davis, Jr., which showed that they had been under treatment for some intestinal trouble repeatedly over a period of several years before the hypertension developed.

Nine of these patients had chronic tonsillitis; many had pyorrhea or carious teeth or evidence of alveolar abscesses; 2 had bronchiectasis; 2 had cholecystitis; 2 had chronic bronchitis; 1 had an aortic stenosis and insufficiency; 1 had a chronic osteomyelitis of the head of the femur of many years' duration, with sinus formation; 3 had diabetes; 2 had simple goiters; 1 had asthma; 2 had hypertrophied prostates, and 1 had a cystitis cystica from catheterization while in coma.

While this series of cases is too small to form a basis for any generalizations, the finding of symptoms of intestinal pathology of a type usually associated with the absorption of toxic substances in over 90 per cent. of the patients is at least worthy of mention. It is also interesting to note that the symptoms of intestinal trouble were present for a long time before there were any symptoms of hypertension.

Two-thirds of the patients in this series were overweight and only 2 or 3 were underweight. Of those underweight, 1 had bronchiectasis and lues; 1 had arthritis deformans, mild and chronic tonsillitis; and 1 had had uremic coma. This, together

with the fact that 63 per cent. of the patients were women, and that all but 2 were over forty, would make it seem that there might be an endocrine factor that was of importance etiologically. However, overeating would also account for the excess weight. Also it is possible to find as many patients of the same age and weight who have had the same type of intestinal trouble clinically, but who have no hypertension.

The most efficacious treatment of hypertension in my experience has been a low protein, low salt diet. High protein diets in which salt is used freely may not aggravate the symptoms, cause the blood-pressure to increase, or cause albuminuria, especially if the diet contains a sufficient quantity of residue to keep the bowels properly regulated without the use of laxatives and cathartics. A real clinical improvement, such as is shown in the cases cited above, where pressures of 200 have dropped 30 to 50 or 60 millimeters and have remained down without medicinal treatment, has been seen only in patients who have been on the low protein, low salt diet. The longer the hypertension has been present, the harder it is to reduce it by dietary or other therapeutic measures. It would seem that the toxic substance giving rise to the hypertension eventually causes permanent changes in the vascular system that cannot be overcome.

It is a well-known fact that a low protein diet which contains an abundance of vegetables and sufficient residue will overcome constipation and probably also cause changes in the intestinal flora. The putrefactive organisms of the intestinal tract do not grow well on protein-free culture-medium, so a low protein diet has a tendency to diminish intestinal putrefaction. This should eliminate certain toxic substances which if absorbed from the intestinal tract might be instrumental in causing hypertension. The addition of lactose in fairly large doses and the administration of buttermilk or, better, Bacillus acidophilus milk will make more of a change in the intestinal flora. Recently this has been tried in a few cases, and the results to date are sufficiently satisfactory to warrant further use of this method.

In arterial hypertension there is an increased tonus in the musculature of the blood-vessels which, if of sufficient duration, will give rise to demonstrable sclerosis. In the early cases it is possible that the muscles of the capillaries may be chiefly involved. This increased tonus is apparently something like a tetanic spasm. Recent work by Luckhardt and others has shown that tetany due to parathyroidectomy can be eliminated in dogs if they are put on a protein-free diet or if they are given sufficient quantities of calcium lactate. Luckhardt is of the opinion that parathyroid tetany is due to the absorption of toxic material from the intestinal tract; that the formation of this toxic substance may be stopped by putting the dogs on a protein-free diet; that the toxic substance may be neutralized by the administration of sufficient quantities of calcium lactate.

In hypertension, in addition to the tetanic condition of the vascular system, cramps in various skeletal muscles are common. It is generally believed that these are due to impaired circulation. Is it not possible that they are tetanic in nature and due to absorption of toxic material from the intestinal tract? If toxemia of intestinal origin has anything to do with arterial hypertension it would seem that the administration of calcium lactate in sufficiently large doses should be of value.

The presence of symptoms of chronic intestinal trouble in 50 of 55 patients with arterial hypertension, which symptoms were present for from one to thirty years before the hypertension was discovered, together with the fact that the most effective treatment of hypertension is the diet that will most readily correct such intestinal trouble, make these observations of interest. A much larger series of cases must be carefully studied before any conclusions can be drawn.

CLINIC OF DR. JESSE R. GERSTLEY

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL[1]

RESHAPING SOME IDEAS OF INFANTILE ECZEMA

The Greatest Errors in Therapy are Those which Emphasize the Treatment of an Individual Symptom to the Detriment of the Whole Child. This Error Has Been Committed too Frequently in the Treatment of Infantile Eczema. From the Standpoint of the Pediatrician the Essential Factor in Any Form of Therapy is to Place the Welfare of the Whole Child Above that of an Individual Symptom. If the Welfare of the Whole Child Demands Treatment which Temporarily May Make the Eczema Worse, that Treatment Should Be Followed. Often, as the Condition of the Whole Child Improves, Obstinate Cases of Eczema Become Milder in Proportion to the Improvement in General Nutrition.

THE patient of this morning presents the following history: He is an only child, three months of age. There is nothing of medical interest in the family or past history. Pregnancy was uneventful. Labor set in at full term and was normal. He was breast fed for two and a half months and then put on the bottle.

Present Complaint.—The mother states that at six weeks of age isolated papules appeared upon his face. These increased in number, alternately getting better and worse, until both cheeks were involved. When the rash first appeared the mother bought some salve at the drug store and applied it intermittently, but this treatment was ineffectual. Some weeks ago she consulted a physician, who diagnosed an eczema due to too much fat in the milk. He advised removing the baby from the breast and placing him on a mixture of skimmed milk. This change of diet resulted in marked improvement of the skin. However, the child became peevish, irritable, lost weight, and did not look well.

About a week ago the mother consulted another physician,

[1] From the Department of Pediatrics.

who ordered the breast feeding resumed. With the change in diet the baby at once gained in weight, became happier and more cheerful. The eczema, however, returned, and is now starting to ooze. The mother is greatly discouraged and comes for advice.

Examination.—The child is somewhat undernourished, but generally healthy looking. Notice especially the blond hair and blue eyes. The cheeks are reddened, infiltrated, covered with pin-point papules, and slightly oozing. The skin of the buttocks is also red and infiltrated. Otherwise the texture of the skin of the whole body is *smooth*, *velvety*, and *delicate*. There is no rickets, no evidence of organic disease, and no abnormality.

Here is a typical case of infantile eczema. Clinically these eczemas are of two types: One starting in earliest infancy covers the hairy region of the head and face with scales and crusts; the other, following the above, covers the cheeks in the second or third month with redness and infiltration. The latter is the type here represented.

This patient serves to illustrate what I believe to be exactly the wrong principle in treatment. Before going directly into this subject, however, let us review for a few moments the ideas of the past ten years.

From the period of beginning bacteriology when Unna sought a specific micro-organism, the conception of eczema has reflected every wave of medical thought. In 1913 the propaganda for vagotonia led to the atropin treatment. With the revival of constitutional syndromes Czerny spoke of the "exudative diathesis." In the newer studies of focal infection tonsils have been implicated and good results reported following their removal. The present tendency to ascribe many symptoms to the phenomenon of allergy has found its echo in treatment, and the idea of hypersensitization to foreign proteins is coming to its own. Meanwhile dermatologists have repeatedly emphasized the importance of local irritation.

During the eight years in which the writer has been interested in the subject the majority of pediatricians have followed the teachings of Adalbert Czerny, who presumed a type of

child specifically predisposed to ezcema, and then attempted to influence the underlying constitution by changes in diet. The term "exudative diathesis" was conceived to describe the anomaly. The factor most influential in affecting this peculiar type of constitution was diet. Studies as to the effect of diet led to the implication of fat, and fat withdrawal became an essential to treatment. Hence, for the last decade the majority of pediatricians and all medical men influenced by them have believed that eczema in infants is due to a peculiar constitution, the so-called "exudative diathesis," and the ideal treatment of this predisposing constitution is a diet low in fat.

However, if one reviews the literature of the last ten years he finds that clinical descriptions of this syndrome vary greatly. Innumerable articles have been written; but with more careful analyses of the literature the definiteness of the picture becomes blurred. No two men have the same idea, or if they do, enough symptoms are included to describe almost any pathologic condition.

Not being definitely satisfied with the situation, it occurred to me that a feasible method of reopening the problem would be to follow various groups of newborn infants and to observe the course and characteristics of those in whom the eczema developed.

After eight years of such observations I am by no means convinced of any definite constitutional anomaly. Eczemas develop in the overweight and underweight, in those with adenopathies and those without, in those with subcutaneous skin flabby and those with it firm. True, the vast majority of children have blond hair and blue eyes, and in all but the involved parts skin of very delicate, fine texture. However, these findings need not signify anything of a deep constitutional nature; they merely suggest a deficiency of pigment.

Regarding the effect of diet, I am by no means convinced that diet in itself is a factor of very great importance. In the first place the absence of many cases showing the syndrome described as "exudative diathesis" would fundamentally invalidate the idea of withdrawal of fat, for fat withdrawal was

devised only to influence the basic diathesis. However, we shall consider diet in more detail later.

In these cases it was observed that these children were especially prone to diaper rashes. Changes in the quality of the stool, the giving of castor oil, a change in the soap used in washing the diaper, diapers wet with urine, all cause erythema and infiltration around the buttocks.

A number of other factors also were of interest. Eczemas are not constant, they come and go, get better and worse not only on different days, but at different periods of the same day. They heal spontaneously some time after the sixth month, and by no means do all last the conventional forty weeks.

For a number of years mothers of eczematous children were questioned carefully as to influences making the eczema better or worse. Answers to these questions have been exceedingly interesting. In number they are legion, and as for analyzing and classifying them one is overwhelmed by their variety.

To the average mother anything making the cheeks red makes the eczema worse, anything making them pale makes it better; anything making the cheeks moist makes the eczema worse, and anything making them dry makes it better. She overlooks completely any fundamental change in the skin itself, but her description of the angriness of the process depends entirely upon the degree of redness and oozing. Eventually it may be seen that three factors are of importance:

A. The factor causing the *eczema itself.*

B. A factor, probably vasomotor instability, which influences blushing and paling of the cheeks.

C. A factor influencing the weeping. Probably this depends upon the amount of fluid in the tissues and so is purely incidental.

A. **The Cause of Eczema.**—In searching for the cause of the eczema itself (the condition characterized by discrete dry papules on a non-infiltrated skin) one might reason as follows:

If an infant with a beautiful velvety skin shows two areas of erythema and infiltration, one on the buttocks and one on the face, the former being due to a host of irritants different in all

children, it is only logical to assume that perhaps irritants are also at work upon the face. This fact assumes importance when one recalls that the only two parts of the baby's skin exposed to irritation are the diaper region and the cheeks, for clothing protects the rest. Surprisingly frequently, a conscientious search often will reveal an irritant long overlooked.

1. Vaselin bought at one drug store always injures; at another, helps. Various ointments do more harm than good. Olive oil at times is harmful, as are some vanishing creams. Perspiration is bad, particularly when mixed with some salve and left for an unlimited time on unwashed cheeks. Interesting in this connection is the report that laborers in ammunition plants working with dyes developed eczema on those parts of the body covered with perspiration. A strong wind as well as a wind carrying dust and dirt is irritating, as is cold air. Indeed, the old family practitioner aptly stated, "November and March are the eczema months." At three months the baby begins to drool and smears saliva all over its cheeks. The nasal secretion of coryza is not a soothing lotion. Exacerbations indignantly attributed by mothers to cod-liver oil or milk too rich in fat may be traced to the irritation of acid stomach contents regurgitated over delicate cheeks. One baby gets worse evenings when his unshaven father comes home and puts the baby's cheeks to his own. Very hot dry rooms in winter irritate and roughen the skin.

2. A factor in connection with the above is called by some *neuropathy*. Some of these children are exceedingly restless and active. They scratch their bodies continuously, particularly when fatigued, and the only exposed part is the cheeks. Possibly an itching skin, a neurodermatitis, is the basis. At any rate, an incipient eczema follows the trauma.

3. Aligned with local irritants, in neglected cases, are *bacteria*. In a very instructive study Lobenstein determined quantitatively that per square centimeter of skin in a normal infant bacteria vary from 246 on the cheeks to 29 on the breast regions. In cases of eczema the findings on the *normal* skin are unchanged, while those in the involved regions are enormously increased—from 3000 in the dry eczema to as high as 40,000 in

some of the moist types. While the day of a specific organism is past, the influences of these secondary invaders must not be overlooked. Thus an attempt to dry weeping cases has an additional impetus.

4. In a few cases, in spite of most searching inquiry, no local cause could be discovered. It occurred to me that, in view of our newer knowledge concerning the effects of light, a factor might here be revealed. Ultraviolet rays of sunlight are known to produce definite changes in the skin: (*a*) Vasodilatation and dermatitis of some duration and (*b*) pigmentation.

Even in adults the hands and face are redder and browner than the covered parts of the body. Again, a skin once affected by these rays reacts with erythema to any irritant much more readily than does a normal skin, even to such general factors as emotion. It was Darwin who recorded that in those tribes much of whose skin is exposed to sunlight emotion might cause blushing of the entire area. Some babies get their first eczema in early spring when taken out of doors for the first time.

Protection against these chemical rays is by deposition of pigment. Areas of vitiligo are especially susceptible to sunlight, and if on the face are easily irritated. Susceptible individuals must wear shields of some sort.

Children come to the world with a deficiency of pigment, some more than others. As eczema seems to occur especially in those of pale, unpigmented skin, the effect of light must not be overlooked, and in this connection one cannot help observing that many eczemas heal spontaneously when blue eyes and blond hair become darker.

Dermatologists have noticed that some peasants at times react to these rays, at times do not. Plant pollens, such as buckwheat, play a rôle, and in seasons when this is in the air it seems to sensitize the skin of susceptible individuals so that the light rays are particularly virulent. Perhaps here we have a clue to the occasional relationship of eczema and asthma in children. The possible effect of light has occurred to others, and Finkelstein recently quotes mild cases of eczema entirely cured by keeping children in a room with red window panes.

B. **Vasomotor Instability.**—The factor of vasomotor instability is one about which we have only clinical evidence. The mechanism associated with blushing seems involved. Some eczemas get worse when near a stove, others if the child is in a temper. With vasodilatation comes increased blood-supply to the cheeks and the eczema looks redder and angrier. Concentration upon this phase of the problem probably led observers to the atropin treatment.

The influence of intercurrent disease probably belongs in this category. Changes in blood distribution result in paling or blushing of the cheeks. The eczema appears better if the blood-supply is drawn to the internal organs, leaving the skin pale. Possibly here lies an explanation of the ascribed relationship of eczema to asthma. It has long been a tradition that eczema and asthma are one; that with the disappearance of the eczema, asthma sets in. The writer is not at all convinced of a relationship other than incidental. A surprising number of cases of eczema never develop the slightest suggestion of asthma. Many children with asthma never give a history of eczema. The suggested relationship may be due to the changed blood-supply, the cheeks becoming pale and the eczema disappearing during an asthmatic attack. Of course, one must not forget that in some cases eczema and asthma may be due to identical causes, as plant pollen.

C. **Oozing.**—In discussing the phenomenon of oozing we are led to the consideration of diet. Probably this plays a part in two ways:

1. Improper diet means improper nutrition of the skin and increased susceptibility to irritants.

2. Diet by affecting nutrition brings to the cheeks either the flush and tissue fluids of health, or the pallor and dryness of undernutrition, and thus affects the oozing of the eczema.

(1) The first is supported by a large number of observations. Contrary to the popular tradition, a large number of patients were in a state of *undernutrition*. These were given absolutely no local therapy and the uninfluenced effect of food observed. With no local treatment other than cleanliness probably one-

third to one-half of the eczemas clear up markedly when proper nutrition of the skin is established following improvement in general nutrition. If the use of food were confined to any procedure at all which benefits general nutrition, many severe cases would be cured.

Undoubtedly some fat, healthy, overfed, breast children seem to suffer from ezcema. In my opinion food here plays its part not by affecting the eczema directly, but in a second way about to be discussed.

(2) As regards the effect of food upon distribution of fluids in the body, it is known that various food combinations, particularly salts or salts and carbohydrates, cause a shift of water content in the body tissues. If a skin abrasion exists, any food causing a sudden increase in tissue fluid will cause oozing. Withdrawal of such food causes depletion of the tissues and drying. Observation of this phenomenon led to Finkelstein's eczema soup—a mixture particularly low in salts. The depletion of the tissues as evidenced by a great drop in weight leads to a paling and drying of the involved areas, but this paling and drying signifies no improvement in the eczema; it too frequently is the reaction of *undernutrition*. If the diet persists, the baby passes on to a state of malnutrition. Resuming a normal diet brings the blood and lymph of health to the cheeks and oozing recurs.

An excellent study by Lederer throws some light on this process. Fluctuations in blood-water from 81 to 83 per cent. depend on change of diet. He noted that eczematous children seem more prone to rapid fluctuations, and incidentally that when there was a sharp rise of blood-water the eczema became moist.

It is not unlikely that the benefit attributed to fat withdrawal is due to these factors. Fat withdrawal seems effective only when *sufficient to cause a loss of weight*. Drying of the tissues and the reaction of undernutrition follows. The same would follow withdrawal of protein, carbohydrate, salt, or water—anything which produces starvation. In the same way anything causing a sudden gain and an increased fluid in the tissues causes a recurrence of the oozing. Feeding fat to an

undernourished child in amounts *insufficient to cause a gain* does not make the eczema worse. In an undernourished child fat even in considerable quantities, as in Czerny's butter-flour mixture, often leads to great *improvement* of the skin by improving general nutrition.

As Lederer in his experimental work incidentally noted that with fluctuations in blood-water the eczema became moist, it occurred to me that perhaps after the blood-water has become adjusted to a new level, oozing would stop. *This is just what happens. Here lies one of the secrets of the dietetic therapy of eczema. If medical men would be just a bit more patient before drawing conclusions they would learn that in an undernourished child, many diets which make the eczema temporarily worse, bringing increased redness and weeping, will, if persisted in, cure the very same eczema by improving general nutrition. After the initial weeping the tissue fluids settle at a new level, the baby gains in weight, and with the improved nutrition the skin slowly heals.*

By no means do I wish dogmatically to assert that all factors have been disclosed. At present there is a tendency to attribute some eczemas to foreign proteins. The work of Shannon and that of O'Keefe is highly suggestive. However, the eczema of infants would be an odd clinical type of protein reaction. Neither in clinical appearance nor in course does it resemble the usual well-known allergy, nor is the demonstration of an individual's susceptibility to foreign protein proof in itself that the eczema is due to this cause. Grulee has reported the case of a child with eczema who was hypersensitive to a particular food, and when getting this would react with typical urticaria. In this one case at any rate two different mechanisms were involved. Possibly foreign protein may cause itching of the skin, which would lead to scratching and eczema on such a basis.

It is interesting to note that some dermatologists have recently considered abandoning the term "eczema." In pathology, course, symptoms, and treatment they see nothing but a dermatitis. They speak of dermatitis of known and unknown etiology. For the present this is probably the most practicable conception.

Treatment.—The treatment must consider the baby himself, the eczema itself, the vasolabile constitution, and the secondary manifestations, as oozing and infection.

As regards the first, the welfare of the whole child *must be considered above any individual group of symptoms*—particularly as most eczemas heal spontaneously after the sixth month. The worst treatment possible is to permit local symptoms to influence the general principles of feeding. The diet must be such as to bring the child to the best possible state of nutrition. *No element of food specifically aids the eczema; no element harms.* By properly directing the feeding, however, one can bring the child to such a state of good nutrition that the well-nourished skin will aid its own recovery. Food should be reduced only in case a child is very definitely being overfed.

As regards the treatment of the disease itself, I prefer to consider eczema simply the result of an unknown or known irritant upon a sensitive, deficiently pigmented skin. One must protect the face from all known irritants, as wind, dust, moisture, and trauma. A different irritant may be concerned in each exacerbation. The face should be *kept clean and, above all, dry.* Perspiration and powder left on unwashed cheeks are not a soothing combination. As for applications I have found nothing better than a lotion of equal parts of glycerin and rose-water, or simply a good oily cold cream. Recently dermatologists have tried with some success in their obstinate cases the following ointment:

Crude coal-tar	2
Zinc oxid	2
Starch	16
Vaselin	16

For a vasolabile constitution nothing definite is known. Possibly bromids are of value, but this is pure hypothesis.

In oozing without infection, remember not to tamper with the diet. The oozing will cease of itself.

In oozing with secondary infection a *temporary* restriction of the diet may be of value not for any effect upon the eczema, but by drying the field and thus leaving it less fertile for bacterial

growth. The application of a mild white precipitate ointment will aid overcoming this complication.

Prophylaxis.—During all these years very little has been written of prophylactic treatment, as if there were no such thing. I believe very definitely that there is a prophylactic treatment of eczema. Once established, a disturbance of nutrition requires much time and skill for its cure; so it is with eczema. Just as the infant welfare idea—"Keep the well baby well"—has reduced infant mortality by incredible figures, so can much eczema be avoided by a slogan, "Keep the well skin well and free from irritants." By such a procedure I believe I have helped abort eczematous manifestations in children who were certainly predisposed. The method is simply a diet to maintain good nutrition and a firm determination not to change this diet and undermine good nutrition for the sake of a few eczematous papules. As for protection from irritants, that differs in each case depending upon the nature of the irritant. After all, the most efficient weapon is a little good common sense.

CLINIC OF DR. JAMES G. CARR

COOK COUNTY HOSPITAL

JAUNDICE

TODAY we want to discuss with you the subject of jaundice. Our first patient, this young colored man, is twenty-five years of age and a laborer by occupation. He was admitted to the hospital on March 21st. He stated that since his discharge from the army in 1918 he has not felt well; sixteen days ago he had a sudden onset of illness, beginning with chills, which were quickly followed by fever and vomiting. A physician who saw him said that his temperature was over 101° F.; the vomiting occurred quite often over a period of several days and was not related to the taking of food. Six days after the chills he noticed the presence of jaundice. His stools have been light in color. Upon admission physical examination was practically negative except for the presence of jaundice, which, as you see, is not intense. His temperature is normal, pulse about 80; the urine on the first examination showed albumin and casts, hyaline and granular, as well as bile and some red blood-cells. The second specimen of urine examined was normal except for the presence of bile. The leukocytes were 9800, the blood chemistry gave practically normal results. The spleen and liver were not palpable. There is no history of hemorrhages, and no evidence of any tendency to such has been brought out in the physical examination. The patient had syphilis while in the army, for which he was treated at that time. He has had no treatment since. His Wassermann is negative and he has not received any arsenicals in over four years.

Infectious jaundice is the diagnosis which seems most logical. The sudden onset of an illness accompanied with the general

symptoms of an infectious process, the marked gastro-intestinal disturbances, the appearance on the sixth day of jaundice with clay-colored stools, with the absence of the characteristic signs of disease of the gall-bladder or the larger ducts, leave us hardly any choice but to accept as the correct diagnosis the condition mentioned.

Not long since we had in our ward another young colored man, who came to the hospital with a history of having been sick for six days. In his case the disease had come on suddenly with severe headache and pains in the back and legs of great intensity. These pains continued for several days, though they were better at the time of his admission. On the second day of his illness he had been seen by a physician who told him that he had a temperature of 103.4° F. and made a diagnosis of "la grippe." There had been one nosebleed, but no other hemorrhage. He complained of no cough or of thoracic or abdominal pain. He had not vomited nor had he been nauseated.

At the time of his admission physical examination revealed the following principal findings: "Eyes negative except for a moderate degree of scleral icterus; mucous membrane of mouth icteric; liver and spleen both palpable; inguinal adenopathy; an absence of abdominal tenderness or rigidity." The urine contained much bile and the stools were light in color. On the day of admission he had a severe epistaxis which continued at intervals through the next day. An examination in the Nose and Throat Department disclosed a small ulcer of the nasal mucous membrane, which was cauterized. Thereafter he had only one recurrence of his nosebleed. Through the first week under our observation he had a daily range of temperature up to about 99.4° F. His blood examination showed Hbg. 70 per cent., erythrocytes 4,250,000, leukocytes 12,700. The Wassermann reaction was negative. The jaundice gradually subsided, and some two weeks after his admission he went home in good condition.

This second case presented a more typical picture of the symptom-complex mentioned, infectious jaundice, which used to be called "Weil's disease," and more recently has been given

the descriptive name, "spirochetosis icterohæmorrhagica." Some seven or eight years ago Inada and his co-workers identified a spirochete as the specific organism of this disease. Since that time Noguchi has discovered a spirochete to be the cause of yellow fever; there is probably a close relationship between the two diseases. Ryle, in his extensive report on infectious jaundice, makes this statement, "Except for its greater severity and the tendency to gastric hemorrhage, yellow fever is clinically the same disease as spirochetosis." This specific form of jaundice has a tendency to occur in epidemics, though epidemic occurrence is by no means invariable; isolated cases are often seen; it is altogether possible that isolated cases are seen and unrecognized. The rat is believed to be the carrier; the infection does not appear to be carried to man by intermediate hosts, as insects, but probably occurs directly through the contamination of food or drink by the excreta of infected rats; the spirochete has been shown to be especially prevalent in the kidneys and urine of infected rats. The disease is believed to be more common in wet seasons and among people who have to live or work in damp places.

Anatomically the brunt of the disease is borne by the liver, which shows degenerative changes of varying degree; in the more severe cases these changes are the destructive alterations commonly seen in acute yellow atrophy; of the other viscera the kidneys and duodenum are the only ones involved with any regularity. The spirochete can rarely be demonstrated after the first week of the disease. Ryle insists that the diagnosis can be made from the clinical picture. He stresses as the important diagnostic symptoms the sudden onset with the subjective symptoms of an acute infection, the pains in the back, and especially the pains in the legs (which latter Ryle considers very significant and mentions the confusion of this condition with trench fever which was sometimes occasioned by this symptom), the continued fever with the supervention of jaundice after five or six days, the tendency to hemorrhage, especially epistaxis and hemoptysis, and suffusion of the eyes.

In the case before you you have already noted the presence

of many of these symptoms, and likewise the absence of some of those listed as important. This patient has had no hemorrhages and does not emphasize the presence of pains in the legs. The second patient whose history I have recounted to you had a more characteristic history; one of his principal complaints was of the pains in the legs; he had nosebleed before his entrance and afterward; yet he did not have the gastric symptoms of which the patient here has complained so much. As we review these 2 cases we feel justified in regarding them both as cases of spirochetosis even though all of the diagnostic symptoms expected are not present; you will learn that diagnosis rarely depends on the presence of all the symptoms and physical findings, which one who describes the disease may regard as "diagnostic." The symptoms lacking were not the same in the 2 cases; the symptoms and physical findings present are such as accord with the diagnosis given, and there are no symptoms present in either case which are incompatible with such a diagnosis. Doubtless, many of you have wondered at the failure on my part to refer to the significance of splenic enlargement in arriving at a diagnosis. In one of the cases under discussion the spleen was readily palpable, in the other it could not be felt. The subject of splenic enlargement has been, thus far, purposely avoided. In the text-books it is usually stated that the spleen is enlarged in this condition, and a palpable spleen is regularly expected by clinicians, yet Ryle says that he did not find this enlargement clinically, and in the cases which went to autopsy the spleen was never found enlarged.

In discussing these cases we recall a third case, a patient who was admitted to the hospital last Thanksgiving Day. This man was brought to the ward in a semicomatose state, muttering unintelligibly, and entirely unable to give satisfactory answers to any questions. From his wife it was learned that the onset of the disease was sudden with chills. Prior to the time of definite onset he had not felt very well; he was depressed and his appetite was not good. With the onset of the acute symptoms he began to vomit and the vomiting continued for several days. He also had abdominal pain, localized in the epigastrium.

There was some ill-defined difficulty of urination; it had been necessary to resort to catheterization the day before he was brought to the hospital. His partially stuporous state had come on the same day. Upon admission the temperature was 99.8° F., pulse 132, and respirations 26. The blood-pressure was 134/80. The urine showed albumin, hyaline and granular casts, and bile. There was a moderate increase in the leukocytes, the count being 13,200. The physical examination was practically without significance; examination of the abdomen was especially unsatisfactory because of the voluntary rigidity which was encountered. A moderate grade of nitrogenous retention was shown by the blood chemistry, the non-protein nitrogen was 55.24, the uric acid 4, and the creatinin 1.85. The provisional diagnosis was made of uremia or encephalitis. The demonstration of the bile in the urine was followed by the definite appearance of jaundice, which led to the diagnosis of infectious jaundice. Shortly after his admission the patient began to improve; he made a steady gain, and two weeks after his entrance the urine was free of bile and of any evidence of nephritis; the jaundice had entirely disappeared. Three weeks after entrance he left the hospital having made a complete recovery.

In this last case we were probably dealing with a more severe type of spirochetosis icterohæmorrhagica than in the other two. A remark of Ryle's in connection with the period of incubation is of interest here; in this last case you will have noted that the sudden onset was preceded by a period in which the patient did not feel well. Ryle says that such premonitory symptoms are not uncommon. The severe and persistent vomiting, the greater prostration, and the mental symptoms all stamped the infection as of more than ordinary virulence. These cases are not rarities; at times, indeed, there has been confusion with meningitis. Though our experience in this hospital has been such as to leave us with the impression that the mortality of the disease is not great, statistics from widely separated countries show a mortality ranging from 7 to 20 per cent. In our case the rapid disappearance of the jaundice, the coincident

recession of the findings of renal irritation, and the prompt recovery of the patient appear to justify the diagnosis of spirochetosis. Doubtless you have observed that these cases under consideration were all practically afebrile while under our care; this has been due to the comparatively late period in the course of the disease at which they came to the hospital. The temperature usually returns to normal about the end of the first week, though there may be a slow defervescence with the maintenance of a slight fever for some days, or there may be recrudescences of the symptoms with exacerbations of fever.

This next patient, also a young man twenty-five years of age, came to the hospital stating that he had not been well for three weeks. His illness began with a sensation of general weakness after a few days of which he was obliged to quit work. By this time his urine had become quite dark and he was annoyed some by itching. He vomited several times during the first few days of his sickness, but had no chill, no fever, and no pain. Three days after he began to feel badly he noticed that his skin was yellow. He has lost 10 pounds in weight. Upon admission his temperature was 98° F., pulse 60, respirations 20. The skin was everywhere icteric to a marked degree. The liver was palpable about a fingerbreadth below the costal margin, but the spleen could not be felt. The urine contained much bile, but no albumin or sugar; the leukocyte count was 9200. Since coming to the hospital he has had a normal temperature. The stools have been "clay colored."

Undoubtedly this case suggests to every one of you the diagnosis of catarrhal jaundice, and such has been accepted by us as the probable diagnosis. The age of the patient, the presence of icterus of the obstructive type, the onset of the icterus with gastro-intestinal symptoms, but without pain, the absence of any febrile symptoms, the failure to demonstrate local findings of cholecystitis or cholelithiasis, and the bradycardia (which was not present in the infectious cases) make up a clinical picture only to be interpreted as catarrhal jaundice. Perhaps this diagnosis should not be made with finality in our own minds

until the patient is well, paradoxic as such a statement seems. However, it is most important not to allow the diagnosis of this benign form of jaundice to blind us to the possibility of some serious underlying disease. Of this there will be some consideration further on.

The next patient will not detain us long. One week ago this man was admitted with a right lower lobe pneumonia. In passing it might be remarked that he was sent to the hospital with the diagnosis of appendicitis, an illustration of the difficulty frequently encountered in the differentiation of pneumonia and appendicitis. Day before yesterday it was discovered that this patient had become quite icteric; since yesterday morning he has had his crisis. Though the icterus is still present, as you see, it is much less distinct today. Jaundice is not a rare complication of pneumonia and is often spoken of as of serious prognostic import. It has not been our experience that this opinion of the very bad prognostic import of jaundice as a complication of pneumonia is justified.

Another patient whom we wish you to see came to the hospital with a minor grade of jaundice, but with other symptoms as his reason for seeking the hospital. Swelling of the abdomen and discomfort in the right upper quadrant have been present for some time, though he is unable to give the exact date of onset. Though there was a distinct icteric tingeing of the sclera and of the oral mucous membrane, the patient stated that he had not been jaundiced. Upon admission there was a marked prominence in the upper abdominal region. The lower edge of the liver extended to the level of the umbilicus; the edge was round and smooth. The spleen was palpable a full handbreadth below the costal arch. The blood-pressure was 114/82. During his stay in the hospital there has been no elevation of temperature. At the time of his entrance to the hospital examination of the blood showed hemoglobin 43 per cent., erythrocytes 3,232,000, leukocytes 2600, with the polymorphonuclears constituting 70 per cent. of the white cells. The Wassermann test gave a 2+ positive reaction. The diagnosis was made of the syphilitic type of Banti's disease and the

patient was put on specific treatment, mercurial inunctions, and potassium iodid without salvarsan. After ten weeks in the hospital, during which time he has been treated almost continuously with the potassium iodid, but intermittently with mercury, the Wassermann is negative and the blood examination shows hemoglobin 70 per cent., erythrocytes 4,800,000, leukocytes 6200. The liver and spleen are greatly enlarged; we cannot convince ourselves that the treatment has made any difference in the size of these organs. In view of the probability, almost certainty, that once outside the hospital the patient will not continue treatment, and the disease will again become progressive, we have advised splenectomy, but the patient has refused operation and desires to go home.

With the demonstration of the next patient we will take up again the subject of catarrhal jaundice and the importance of care in accepting it as a final diagnosis. This man was in the hospital for some two weeks about two months ago. At that time he stated that he had felt well until some five weeks previously. His illness began with pains in the abdomen, which he ascribed to "gas." These pains were located across the lower abdomen and usually came on two to three hours after eating. An enema would relieve them. Simultaneously, he had had "indigestion," a brassy taste in his mouth, discomfort after eating, and an inability to eat greasy foods because of greasy, sour eructations. At times he felt as if food were held in the esophagus. Jaundice began about ten days before his first entrance into the hospital. This steadily became more intense; after its onset he noticed dizziness and drowsiness. He vomited only once, and then the vomiting occurred after taking ipecac; he had felt nauseated, especially after meals. He had a carbuncle last fall; when this was at its height he had chills and fever. When he first came to the hospital a second carbuncle was developing. He has been constipated for three or four years; this became more severe with the progress of his present sickness. He gave a history of having had both gonorrhea and syphilis years ago. Formerly he drank to excess and still uses tobacco beyond the limits of moderation. His appetite had been

good. He thinks he had passed "clay-colored" stools for months before his admission.

When first admitted the temperature was 98.8° F., pulse 78, respiration 20. The blood-pressure was 88/60. The scleræ were markedly icteric. All his teeth except two are missing, and the two present are crowned. The lungs are normal. A rough systolic murmur was heard at the apex and transmitted to the axilla. The liver could be felt, but not the spleen. The skin showed jaundice of moderate intensity. The urine contained bile; albumin and sugar were not found. The Wassermann reaction was negative. The leukocyte count was 6000. Examination of the stomach contents following an Ewald meal gave these results: 300 c.c. were recovered, the total acidity was 76, and the free acidity 40. The report of the roentgenologist read: "Completeness of curvatures, duodenal bulb of satisfactory outline, and active peristalsis upon screen examination render the examination negative for intrinsic lesions of the stomach or duodenum." After two weeks in the hospital the patient went home at his own request. At the time of his discharge it was noted that the jaundice seemed to be diminishing in intensity. The diagnosis written on the discharge slip was "catarrhal jaundice."

Within less than five weeks he returned; during that time the jaundice had grown worse; there was a steady loss of weight and strength. Upon his second admission he was sent to our ward again, but in view of the previous history, the long duration of the jaundice, its increasing intensity, and the patient's progressive loss of strength and weight, he was promptly transferred to the surgical service for an exploratory operation. At this time the hemoglobin was 45 per cent., the erythrocytes were 2,190,000, and the leukocytes 9600. The liver was three fingerbreadths below the costal margin; it was not tender. Below the liver, at the level of the umbilicus, a palpable rounded mass was felt, which was taken to be the gall-bladder.

At operation the gall-bladder was found distended, and about the ampulla of Vater was found a firm, hard mass the size of an English walnut. A cholecystoduodenostomy was done.

On the fourth day after operation a bile-stained stool was passed. As you see, he is making excellent progress toward recovery.

From the diagnostic standpoint this case is of unusual interest. The incorrect diagnosis of catarrhal jaundice under which he was allowed to leave the hospital as a non-surgical case points its own moral. After five weeks of progressive physical failure he returned for operation, which had to be done under circumstances far less favorable than might have been the case. The outcome has been as happy as could have been expected (for malignancy was really the most likely diagnostic alternative to catarrhal jaundice), but we cannot escape the feeling that the gravity of the operation was needlessly increased by delay. In a patient past middle life the diagnosis of catarrhal jaundice is very likely to be incorrect; occasional cases occur, but too often the jaundice of insidious onset in the individual beyond forty is the sign of malignancy. The valid objection to such a diagnosis in the class of patients mentioned is based on the facts of psychology rather than of medicine, in a stricter sense. Once we reach a conclusion and make a diagnosis, we are too prone to close our minds to the possibility that we may be wrong. The question of diagnosis being settled, it is natural and easy to explain everything from that standpoint. It is far better to keep before our minds that "we do not know," that the cause of jaundice in older people is not explained (it is rather hidden) by the diagnosis of "catarrhal jaundice," and that this diagnosis under these circumstances is only acceptable after the patient has recovered. Neither is the subject one of purely academic interest. It is true that the probable cause of jaundice, characterized as we have described, is malignancy, and all too often neither the physician nor the surgeon can offer a cure. Yet, at the least, the surgeon may often relieve annoying symptoms, such as the intolerable pruritus, make living more comfortable, and prolong life while reducing suffering, and, at the best, it does happen that the icterus has resulted from impaction of a stone, even though typical symptoms are not recounted in the history or from some chronic inflammatory process in or about the ducts, either of which conditions may be perma-

nently relieved by a successful operation. Hard-and-fast rules cannot be established; yet we are sure that after seven weeks of jaundice it was not wise from a diagnostic standpoint to dismiss a man of fifty-three as a sufferer from catarrhal jaundice.

Before leaving the discussion of this case our attention ought to be called to the results of the gastric analysis. You will recall that the free acid and the total acid were both high, at the upper limits of normal. The question of acidity in cases of malignancy is interesting and important. Often we hear the opinion expressed that the presence of free hydrochloric acid is incompatible with carcinoma of the stomach; our experience has taught us that an achylia is not an invariable accompaniment of gastric carcinoma; though high acid values are rare under such circumstances, normal values may be found, and free acid in less than normal concentration is not very uncommon. In connection with carcinomata about the stomach, in the liver, the duodenum, or the pancreas it is assumed that the finding of gastric contents with normal acid values is evidence against the existence of cancer; in general, this is probably correct, but the findings in this case prove that the presence of free and total acid in normal quantities does not exclude the possibility of cancer in some organ closely connected with the stomach anatomically and physiologically.

There is this one other patient whom we wish you to see this morning. This man is fifty-three years old, a baker by occupation. He was admitted to the ward March 6th, with a history of having been far from well for eight months, though not until quite recently has he considered himself actually sick. At first, some eight months ago, he had a lazy feeling, so that he would have to lie down in the afternoon. Later he noticed a yellow color, which first appeared in the whites of his eyes and afterward became apparent in the skin. Since its first appearance he has not been entirely free from icterus, though it has varied in intensity from time to time. He has been annoyed excessively by pruritus. Five days ago he noticed swelling of the feet, followed by swelling of the legs and thighs. Swelling of the abdomen and scrotum have developed since.

Lately he has been short of breath on exertion. He has had no abdominal pain and no gastric distress except some from gaseous distention. There has been no vomiting. The stools are yellow.

He has used beer to excess and whisky to a considerable extent. He also uses tobacco. He had pneumonia at twenty-eight and malaria at sixteen years of age. He has had gonorrhea and syphilis. Seventeen years ago he had an attack of jaundice which lasted about a month.

Physical examination shows a distinct jaundice. The teeth are carious, with much pyorrhea. The lungs are negative. There is a systolic murmur heard at the apex. The abdomen is markedly distended; this distention is largely due to the presence of fluid. The liver is tender; a hard sharp margin can be felt a little way below the costal arch. Internal and external hemorrhoids are present. Both lower extremities and the scrotum are very edematous. The reflexes are normal. Two days after admission 2200 c.c. of fluid were withdrawn from the abdomen; the specific gravity of this fluid was 1005. Since the tapping the spleen can be readily felt. Except for the presence of bile the urine is negative. The blood shows erythrocytes 3,150,000 and leukocytes 6400. The Wassermann test has not been made, as the single specimen thus far withdrawn proved to be anticomplimentary. The stools, though lighter in color than is normal, give a positive test for bile.

The diagnosis has been the subject of much discussion. Influenced very largely by the patient's emphatic statement that the swelling began in the legs, spread upward, and appeared last of all in the abdomen, the house physician inclined to a diagnosis of cardiac decompensation; his opinion was further supported by the history of dyspnea and the presence of a cardiac murmur. Yet such an explanation leaves one important symptom unexplained; the diagnosis of cardiac insufficiency does not account for such jaundice as you see here, and the jaundice preceded the edema by many months. The obvious diagnosis is atrophic cirrhosis. The sequence of onset of the edema and ascites (exactly the reverse of that expected with portal obstruction) may be regarded as evidence against

the diagnosis of cirrhosis, yet it is less decisive against the diagnosis of cirrhosis than is the degree of jaundice against the diagnosis of a simple cardiac failure. I recall a case of marked edema of both lower extremities, with ascites, associated with a large gumma of the left lobe of the liver. With the recession of the tumor under treatment the edema also disappeared. Syphilis is characterized by its multiformity; syphilis, cirrhosis with gumma, or gumma with a greatly thickened hepatic capsule, might explain the symptoms here. There is still the further possibility that the patient has failed to observe a gradually increasing distention of his abdomen until the rapid onset of the edema elsewhere. Perhaps the symptoms now present are the result of the combined factors—cardiac and hepatic syphilis. Looked at in this way the abdominal enlargement may have precipitated cardiac failure partly by mechanical means and partly because of the intoxication due to hepatic insufficiency.

However, one is compelled to admit that the marked ascites, the fluid with a specific gravity of 1005, the hemorrhoids, the enlarged liver and spleen (rarely is the spleen palpable as the result of cardiac decompensation), and the jaundice itself all stamp this case as essentially hepatic. If cirrhosis, of what type? Jaundice as deep as this is unusual in atrophic cirrhosis; what you see here is "jaundice," not the subicteric tint so common in patients afflicted with "hob-nail" liver. The liver of atrophic cirrhosis is not often as large as this liver is at a time when the disease has reached the stage of advanced ascites. Finally, there is a definite history of syphilis. Though we may not prove successful in getting a satisfactory Wassermann test, our feeling is that until the therapeutic test has been tried the probability of hepatic syphilis cannot be excluded. He will be given potassium iodid; if with increasing doses this treatment fails to produce improvement, we will be content to accept the diagnosis of atrophic cirrhosis, characterized in this instance by an unusually intense jaundice.

Before leaving the question of diagnosis, we ought, at least, to give our reasons for practically ignoring the possibility of carcinoma. Having emphasized the probability of cancer in

patients beyond forty, who present themselves with a history of jaundice coming on insidiously and becoming steadily more intense, we could hardly be excused for the failure to give to you the reasons for our belief that this case is not one of carcinoma. The reasons may be listed thus: (1) If cancer were responsible for jaundice of several months' duration, we would expect his general physical condition to be much worse than it is; (2) though there is a history of jaundice persisting through several months, it is not yet distinctly obstructive in character; the icterus is evidently of intrahepatic origin rather than of obstruction in the larger ducts; of course, jaundice of this type might be the result of carcinoma in the liver itself, whether primary or secondary to some remote primary source, but this man is not sick enough; a diffuse cancerous infiltration of the liver causing icterus without producing obstruction of the larger ducts cannot be accepted as a plausible explanation in this case where our patient has been sick eight months, yet is not emaciated and of a generally pitiable appearance; (3) the physical examination of the liver reveals a smooth edge and surface; neither is the consistency as hard as that which we find so commonly in hepatic carcinoma; (4) there is no evidence of any primary disease; (5) the fluid is a transudate, a possible finding where the ascites is the result of carcinomatous obstruction of the larger ducts, but not likely after so many months of jaundice.

The subject of jaundice has not been covered exhaustively; no attempt has been made to discuss with you theories of the etiology of icterus, and no classification of the conditions characterized by this symptom has been offered. Rather we have presented to you some cases in which the icterus is a prominent symptom in much the same haphazard way as they will come to you in practice. Particular attention has been directed to the subject of infectious jaundice and to the importance of careful differentiation of the conditions presenting symptoms suggestive of catarrhal jaundice.

Outcome of the Last Case.—A few days after the clinic was given the patient developed a cellulitis of the right lumbar region following an abrasion of the skin caused by scratching.

Two days thereafter he had a typical facial erysipelas and was transferred to the contagious service. After eight days he was again sent to our ward with marked ascites and an abscess of the right side at the site of the cellulitis mentioned. Abdominal paracentesis was done first as the patient was complaining so bitterly of the distention. He was then sent to the surgical service and a large abscess was opened; the patient's general condition was already bad, as it had been since the beginning of the erysipelas, and he died on the third day subsequent to the operation.

Autopsy showed: "Extensive cellulitis of the right side with recent surgical incision; bilateral interlobar fibrous pleuritis; hypertrophic pulmonary osteo-arthropathy; sclerosis of the coronary arteries; sclerosis of the aorta with ulceration and calcification (senile plus syphilitic); ascites; fibrous thickening of the hepatic capsule; cirrhosis of the liver with biliary staining; interstitial pancreatitis. The cirrhosis was not syphilitic. Differential stains of the liver tissue revealed the presence of hemosiderin, suggesting the presence of hemachromatosis."

TUBERCULOSIS OF THE SEROUS MEMBRANES

At this time of the year respiratory infections are frequent, and much of our work in the wards is the differential diagnosis of pulmonary and pleural conditions. Certain phases of these problems will be developed in the discussion of this first case.

This man is forty-five years old and a teamster by occupation. Upon admission he stated that he had been sick two weeks; that his disease had come on slowly, the first symptom being loss of appetite; this was followed by gradually increasing shortness of breath. He began to cough, which annoyed him especially at night; this was productive of a thick whitish sputum, but was unaccompanied by pain in the chest. There was no history of headache, vomiting, abdominal pain, edema, or night-sweats. He had felt feverish at times. His past history was negative so far as sickness was concerned. His mother had died of tuberculosis at forty-five. He uses tobacco, but denies the use of alcohol.

Physical examination at that time showed a well-developed, well-nourished man with some respiratory embarrassment. The temperature was 99.8° F., pulse 120, respirations 24. The blood-pressure was 100/80. The eyes were negative; the teeth were carious and pyorrhea was present. Examination of the chest revealed some bulging of the right side near the base. The right side was and still is almost entirely immobile. There was dulness over the entire lower half of the right side, and over this dull area the fremitus was decreased, and the voice and breath sounds were distant. A pleural friction-rub was heard near the base. Abdominal examination was negative. The reflexes were normal.

Because of the evident respiratory embarrassment the chest was tapped on the day of admission and about 1500 c.c. of clear straw-colored fluid withdrawn. The specific gravity of this fluid was 1016. For three days thereafter the temperature was below 100° F.; then there was a rise, and through a period of

about ten days the temperature reached 101° F. or above every day; during this febrile reaction the patient appeared quite sick, and the question as to the presence of pneumonia was considered. For the past four days there has been no fever. The pulse has varied from 100 to 120 and the respirations have been above 30 a large part of the time, but are now 28. Yesterday the house physician made these notes: "The physical findings of hydrothorax are present; there is flatness up to the level of the fourth dorsal spine; cavity findings are determined just above the nipple."

On March 1st (ten days after the aspiration) a report from the roentgenologist read: "Hydro- or pyopneumothorax is seen with fluid level at upper border of second right costal cartilage apparently extending from median to lateral and anterior and posterior chest wall. However, border of collapsed lung is not seen and apex is less clear than the space immediately above the fluid level. Radiolucence of the opposite side of the chest is fair for an individual of designated age."

If we review the history and physical findings as they have been read to you, we find a history of an illness of insidious onset with shortness of breath and cough. The examination has brought out the physical signs of fluid in the right side of the chest; the signs present are dulness (flatness) on percussion, impaired mobility of the right side, greatly diminished tactile fremitus over the area of flatness, distant voice and breath sounds over the same area, and the absence of Litten's sign on the right side. It might be remarked in passing that such simple and valuable signs as the absence of Litten's phenomenon are too often ignored nowadays; this evidence of the failure of movement of the diaphragm is always significant.

Not much consideration was given, in making our diagnosis, to the question of a probable pneumonia. The history as stated mentioned neither the characteristic onset nor course of lobar pneumonia, while the physical findings were clearly those of fluid. The diagnostic problems were the determination of the character of the fluid and the demonstration of the nature of the primary infection. First of all we had to exclude empy-

ema, and that was done by the aspiration; the fluid was serous, not purulent.

While the question as to the presence of pus was easily settled, the diagnosis from an etiologic standpoint needs more discussion; especially since the house physician neglected to make a differential count of the cells found in the fluid. You know that most of these serous effusions occurring with the symptoms of a moderately severe infection are tuberculous in nature, yet simple non-specific pleurisies do occur, and to the patient it is a matter of first importance whether his pleurisy is tuberculous or not. Lacking the demonstration which might have come from a cell count, we must depend on more general data. The insidious onset without pain, without even thoracic symptomatology at the beginning, the persistently low leukocyte count (three counts have each shown 6200 leukocytes), and the history of his mother's death from tuberculosis are the essential diagnostic features in the case. The evidence in favor of tuberculosis as the correct diagnosis is not very imposing, especially since the roentgenologist's report adds no further evidence in support of such a diagnosis, yet we believe that, in all probability, this is a case of tuberculous pleurisy. To the few facts adduced above we can add the demonstration of a fluid of an inflammatory origin and the important further fact that most of the primary pleurisies are tuberculous; the evidence at hand assumes great importance in the presence of this last fact. If guinea-pig inoculation had been done we might have had positive proof of tuberculosis by this time.

Another interesting feature of the case arises in connection with the question as to the advisability of aspiration in these cases suspected of being pleural tuberculosis. In general, aspiration of tuberculous pleurisies is not good practice, while activity of the process is indicated by the presence of fever; the mere fact of the presence of fluid is not an indication for its removal; rather the presence of the fluid may be regarded in itself as an indication to leave it alone. The expression of opinion concerning the possible influence of the effusion in furthering a cure may excite controversy which would serve no

good purpose here; but it is a fact that the removal of the effusion while the patient is febrile has often been followed by an exacerbation of the symptoms and by diffuse pulmonary tuberculosis, eventually hastening the patient's death. Unless the mechanical indication (respiratory and circulatory embarrassment due to the accumulation of much fluid) is present, these cases had better not be tapped. Added to the danger of dissemination of the disease is the susceptibility to secondary infections which these patients show, as a result of which, after two or three tappings, one is likely to find that a fluid, previously serous, has become purulent. In this particular case the aspiration was done because of the patient's great respiratory distress. Doubtless it was the proper procedure, yet you have surely noted that the removal of the fluid was followed in a few days by an exacerbation of symptoms and by a febrile reaction which was quite marked for a period of two weeks. The mechanical indication, as we have called it, may justify aspiration while the febrile symptoms are pronounced; this indication is based on the presence of circulatory and cardiac embarrassment as manifested by dyspnea and cyanosis. The physician must be the judge in each case as to the proper practice; whether or not to aspirate must be determined in the light of the facts offered by the particular case, but the danger inherent in the aspiration of active tuberculous pleurisy must not be overlooked, and it is right that we should here emphasize to you the possibility that removal of the fluid may cause an aggravation of the disease of such virulence that its further progress may never be stopped. Nothing has been said about the removal of small amounts of fluid for diagnostic purposes; even these small quantities had better not be withdrawn unless an examination of the fluid will be of real help in making the diagnosis, but the withdrawal of small amounts is not fraught with the danger associated with the removal of all, or nearly all, the effusion.

Now, after more than four weeks in the hospital, our patient is showing some definite improvement, especially as concerns his general condition. The evidence of fluid is still present;

indeed, it is so well marked that we are again considering the advisability of aspiration. You recall that a week after the first tapping the *x*-ray revealed a large effusion; the physical signs of such an effusion are readily demonstrable today. An interesting notation has been made on the chart by the house physician, remarking the physical signs of fluid on the right side and the presence of "cavity findings just above the right nipple." As we demonstrate the findings referred to in this last phrase, you hear a hyperresonant or tympanitic note on percussion; the breath and voice sounds are exaggerated, but are not bronchial in character; some moist râles are heard over this area. The interpretation of these findings as evidence of a cavity is probably incorrect; it is more likely that the peculiar note obtained on percussion is the well-known Skoda's resonance, and the other findings are such as are usually found with this characteristic form of resonance. Skoda's resonance, as you well know (but will sometimes forget), is often found over the area of the collapsed lung above the level of the fluid and is due to the physical changes in the compressed and moderately edematous lung; it is a valuable bit of additional evidence in favor of the diagnosis of fluid in questionable cases.

The prognosis in these cases of pleural tuberculosis is, in general, quite good. With proper treatment, which means the usual treatment for tuberculosis, many of these patients recover entirely; of course this implies that subsequent to the improvement the patient finds an occupation and living conditions which are satisfactory. The prognosis becomes more dubious if other serous membranes are involved, which unfortunately often happens. A well-localized tuberculosis is certainly much more amenable to treatment than one widely scattered. In this case there are several reasons why we need to be more guarded in our prognosis; the general appearance of this patient is that of a seriously sick man; there is a family history of tuberculosis; of far more importance in estimating this man's chances is the fact that he entered the hospital with a very large effusion which has quickly reaccumulated after aspiration, and for some six weeks we know that his cardiac and respiratory functions

have been subjected to excessive strain; these factors distinctly modify the prognosis for the worse. It will probably be necessary to aspirate again; unless there soon appears some evidence that the effusion is diminishing, we will tap again in a few days. If, after another aspiration, the patient remains afebrile and rapid reaccumulation does not occur, the outlook will be more hopeful; in the absence of such encouraging developments we must continue to regard the prospects for eventual recovery as far from promising.

Outcome.—For six days after he was seen in clinic the patient had a normal temperature. Then the chest was aspirated and 1600 c.c. of a clear, straw-colored fluid was withdrawn. After the aspiration the patient expressed himself as feeling much better. Through a period of almost five weeks the temperature continued practically normal; only twice were readings above 99° F. recorded. The patient was up and around the ward and was regarded as in a fair way to get well. He was anxious to go home. The daily records made by the nurses read, quite regularly, "Slept well and comfortable, condition good" until May 9th. At this time he again complained of dyspnea; on the night of the eleventh he could not stay in bed; the nurse's report stated that he found the "bed intolerable" and spent the night in a chair. On the preceding day it had been noted that fluid was present in both sides of the chest, "the right chest full of fluid, the heart displaced to the left," and "the chest to be aspirated." On the 12th aspiration was undertaken; twice the needle was inserted without result; on the third attempt about 30 c.c. of clear fluid was withdrawn, but no more could be obtained. On the 13th the *x*-ray report read: "Hydro- or pneumothorax is seen in right lung area with fluid level near lower border of first costal cartilage with much collapse of lung. The lower half of left lung area shows dense shadow with upper border extending outward and slightly upward from lateral border of heart, suggesting fluid or pleural thickening of much less extent. Remainder of lung on this side is somewhat lacking in radiolucence, possibly in part due to compression, but with somewhat nodular shadows throughout."

The patient's general condition was very poor. Since the failure of the aspiration on the 12th, any further attempt to remove fluid was deemed inadvisable. In a few other cases with a similar course we have found at autopsy massive exudates in the pleura or pericardium, fibrinous in character, even as much as ½ inch in thickness, and we believed that a similar condition had developed here. On May 17th the patient expired.

Autopsy: "Marked bilateral hydrothorax, fibrocaseous tuberculosis of the parietal and visceral pleura; marked thickening

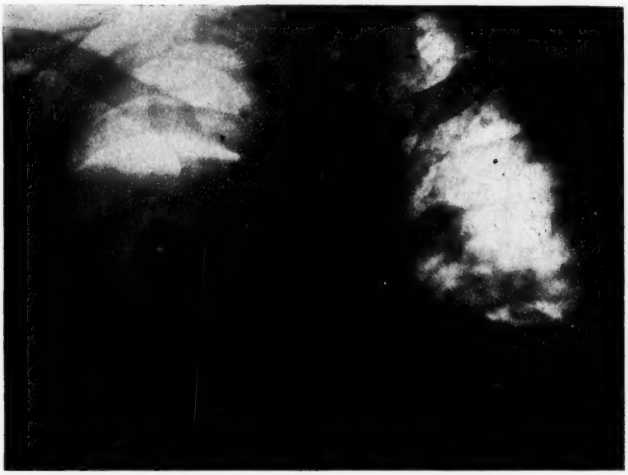

Fig. 91.—*x*-Ray of chest of first patient taken May 13th, four days before his death.

of pleura of right side, with comparatively little fluid; old caseous nodular tuberculosis of the left apex; fibrocaseous tuberculosis of the tracheobronchial lymph-glands; hyperemia and edema of both lungs; compression of right lung; passive hyperemia of liver and kidneys; chronic diffuse nephritis; left pyonephrosis."

It is possible that life might have been prolonged a little while if, instead of the right, the left side had been tapped at the time of the last attempt to aspirate. On the left side the pleural exudate was still fluid; there might be a hint for treatment here;

in such a case of long duration it may be better practice to aspirate the side more recently involved if, eventually, both sides show the evidence of fluid, but the accompanying *x*-ray plate is proof that no great relief could have been expected in this case, even though some fluid had been removed from the left side.

Our next patient shows a slightly different form of tuberculosis, essentially the same anatomically. This man is thirty-six years of age, a Pullman porter by occupation. He came to the hospital with the history of having been sick about three weeks. His illness began with a cold; he had some cough. Within a short time he noticed swelling of the abdomen. About the same time he began to be short of breath. He had no gastro-intestinal symptoms. There was a history of a previous attack of typhoid some years ago. He has also had mumps. He has had gonorrhea, but is not sure about syphilis. He uses tobacco to excess, but denies the use of alcohol. His family history is without significance.

Physical Examination.—The eyes are slightly irregular and do not react to light; teeth are in fairly good shape; there is some roughened breathing about the right hilus and a few moist râles are heard at the same place; fine râles are heard over both apices and a friction-rub, pleural, is found just to the outside of the cardiac area; the heart is negative except for its rapidity; the abdomen is distended, with shifting dulness and a definite fluid wave; the liver and spleen are not palpable; the extremities, genitalia, and rectum are negative; the reflexes are normal, the temperature at that time was 104° F., pulse 136, respirations 44; the blood-pressure, 124/84, the leukocytes numbered 5100.

Six days later it was noted on the chart that the patient was feeling well. His temperature has varied from 100.2° to 104° F., and has been up to 103° F. at least every day; his pulse runs about 120 and his respirations are above 30 all the time. After another six days it was noted that "the breath and voice sounds are increased over the left upper lobe posteriorly"; the abdomen

was greatly distended. On the tenth day a negative Widal was reported. On the twenty-first day the roentgenologist's report read: "The apices were fair; the diaphragm shadows on both sides were high. The diaphragm on the left showed concave instead of convex upper border. Mobility of both was reduced. In the prone position the left lung area was slightly less clear

Fig. 92.—*x*-Ray of chest of second patient taken twenty-six days after his admission to the hospital.

than the right, as shown upon plates, probably from dissemination of small amount of fluid causing alteration in normal outline of left diaphragm, as noted in erect position. Presence of pulmonary tuberculosis is not determined." On the twenty-sixth day the record reads, "Evidence of fluid in both sides of the chest." The Wassermann reaction was negative. Eight

examinations of the urine have been negative, save for one examination, which showed a few hyaline casts.

This man, then, has been sick for three weeks with shortness of breath and swelling of the abdomen, following a cold, and enters the hospital with a temperature of 104° F. and a pulse-rate about 140; at the time of his admission the evidence of ascites was present. Yet, in spite of these very definite findings, he expressed himself as feeling very well; he sought relief from the objective symptom, the abdominal swelling, rather than of any subjective symptoms. The essential points in the history and physical findings were these: The insidious onset of a disease bearing the stamp of an infection with high temperature, rapid pulse, ascites, and a low leukocyte count. To these were added minor signs of pulmonary and pleural involvement. This group of symptoms suggested at once the presence of a peritoneal tuberculosis; indeed, the demonstration of the ascites lacked only the positive findings which might be expected from the examination of the ascitic fluid to make the presumptive diagnosis a certain one. Yet the simple diagnosis of tuberculosis of the peritoneum is not satisfactory in the presence of such a high fever and a rapid pulse; in other words, in the presence of such markedly acute infection. From the start we had in mind the possibility of a miliary process involving the serous membrane of more than one cavity. The presence of minor signs of tuberculosis in the lungs is not an unusual finding in connection with peritoneal tuberculosis, but the later evidence of fluid as shown by the *x*-ray and by the physical signs of fluid in both sides of the chest adds very much to the probability of a multiple serous tuberculosis, in this case miliary in type.

Tuberculosis of the serous membrane is not an uncommon disease among the patients whom we see here in the County Hospital, at least 6 or 8 will be seen on our own service in the course of a year. The condition is far more frequent in the colored people; as is well known, these people are especially susceptible to tuberculosis; probably serous tuberculosis is proportionately much more common among them than the usual varieties of the infection.

The condition may begin insidiously or it may manifest itself as an acute pleurisy or pericarditis; peritoneal involvement causes distention or pain with gradually increasing ascites; a case with an abrupt onset I have never seen, though we are occasionally called upon to differentiate, in persons seen for the first time, acute abdominal symptoms in the course of peritoneal tuberculosis from a true intestinal obstruction. I use the word "true" because tuberculous peritonitis may cause symptoms like those of obstruction which disappear in a day or two and remain unexplained; the great amount of productive inflammation seen in these cases might excuse one for anticipating actual obstruction as a frequent event, though I do not believe that such is the case; I do not recall a single case of the kind. Now and then we see cases of primary pericarditis without other etiology in persons whose general appearance is that of tuberculosis; if evidence of tuberculous infection elsewhere is discovered, active or latent, we feel certain that this pericardial inflammation means the beginning of a multiple serous tuberculous serositis; if no focus can be found, the suspicion is strong that the local process may be the first open manifestation of an active tuberculosis.

Anatomically, the disease is characterized by a disseminated tuberculosis of the serous membranes, pleura, pericardium, peritoneum, or meninges; meningitis is uniformly fatal. Meningitis is a common terminal stage in all forms of tuberculosis and is by no means confined to the type under discussion; in fact, in my own experience, most of the patients with multiple serositis have shown no meningitis, but have died of cardiac embarrassment due to the mechanical effect of large exudates, pleural or pericardial, or of the infection itself. Multiple serous tuberculosis really appears in two quite distinct types, considered anatomically, and these two types can be distinguished clinically in most cases. In the one type we find disseminated miliary tubercles with fluid free in the cavity; these cases run a rapid course. In the other the fibrinous exudate is marked, the serous coats may be enormously thickened, and in the peritoneum many adhesions may be found with pockets of fluid;

tapping such an ascites is a serious business. Perhaps it would be more correct to regard these two types as merely two stages in the development of the one disease, and such is doubtless the correct conception; yet clinically the differences are real and of value in prognosis. The virulence of the infection or the feebleness of the patient's resistance may be measured by the clinical signs of one or the other type of disease; if the course is rapid and acute, we will probably find the miliary type; if it is slow, the progress marked by remissions, and the reaccumulation of large amounts of fluid, we may be sure that the autopsy will show thick fibrinous exudate with fibrocaseous tuberculosis.

The symptoms of onset, the diagnostic symptoms, and the course of the disease (particularly of the acute variety) are very well illustrated by the case before us. There is the general symptomatology of an infection with varying degrees of virulence, without a leukocytosis. With primary peritoneal involvement there is abdominal distention due partly to ascites and partly to the gaseous distention, which is probably the result of the general intestinal atony, and there may be pain, though, as you see in this case, the latter is not invariable; with primary pleurisy or pericarditis the symptoms of onset are like those of any pleurisy or pericarditis. There is likely to be a history of an old focus of tuberculosis or a family history of the disease. Frequently the demonstration of pulmonary involvement clinches the diagnosis. The fluid is of high specific gravity, often hemorrhagic, and contains a predominance of lymphocytes. The proof that the disease has affected more than one serous cavity warrants the diagnosis of multiple serous tuberculosis. The course of the disease may be acute, as this one is; the fever is high, the pulse rapid, and evidence of the spread of the process quickly appears; or the course is chronic, with remissions, during which much improvement is apparent, but with steady progress toward the fatal termination; the fever is not so high, the pulse less rapid (at least until the late stages), and the general loss of weight and strength more gradual.

About prognosis you have already drawn your own conclusions; in the acute form the outlook is very bad; the course of

the disease is likely to be less than two months. Prognosis is much more difficult in the chronic form so far as determining the duration of the disease is concerned, yet much more hopeful because many of these patients will live for years and oftentimes become fairly well over rather long periods of time. In all these cases, even those which appear least virulent, the hope of recovery is almost sure to be illusory. As for treatment, the subject may be dismissed briefly. The general principles of the treatment of tuberculosis must be observed strictly. From time to time, as the indication arises, large accumulations of fluid must be removed.

Outcome.—As was anticipated, no improvement in the condition of the patient took place; the general symptoms of a severe general infection persisted, though no great change was apparent for three weeks; then a sudden aggravation of the symptoms was noted, especially of the abdominal distention. The patient became very weak. Within twenty-four hours death occurred. The rapid increase of the abdominal distention was regarded as largely due to gaseous distention rather than to a rapid accumulation of fluid. Gaseous distention is apt to be a prominent feature in cases of peritoneal tuberculosis and is the source of great distress to the patient. Moreover, it is hard to control.

The autopsy report reads: "Generalized tuberculous peritonitis (miliary); moderate hydroperitoneum; marked gaseous distention of the small and large bowel; moderate bilateral hydrothorax; hyperemia and edema of both lungs; marked fatty changes of the liver; moderate chronic diffuse nephritis."

CLINIC OF DR. ARTHUR F. BYFIELD

COOK COUNTY HOSPITAL

MEDICAL EMERGENCIES

CERTAIN definitions and restrictions are called for first of all. By "medical emergencies" we have reference, of course, to emergencies that confront the internist. What is an emergency? Webster says that an emergency is "any event or occasional combination of circumstances which calls for immediate action or remedy; pressing necessity; exigency." As a generally applicable definition this cannot perhaps be improved upon, yet for the purposes of our particular theme it demands certain modifications and interpretations.

We believe that if a physician were called upon to give an immediate definition of a medical emergency his mind would involuntarily picture a condition demanding quick, decisive medical treatment. Our experience indicates that this point of view represents only a small fraction of the truth. A medical emergency, we would say, calls far more for prompt and accurate diagnosis than for therapy. The percentage of cases met by the internist demanding immediate treatment to save life or prevent irreparable damage is relatively small; the percentage of cases demanding immediate or early diagnosis is, on the other hand, enormous.

Several years ago we published a Clinic in the Medical Clinics of North America which we called "Important Causes of Errors in Diagnosis." In this paper we named six common causes of diagnostic error, as follows: 1. Incomplete or incorrect case-history taking. 2. Incomplete examination of the patient. 3. Ignorance of certain pathologic complexes. 4. Failure to explain

the atypical in a case, or conversely, failure to build up a diagnosis upon the typical. 5. A plus of laboratory detail and of ultramodern methods at the expense of sound judgment and good sense. 6. The riding of a hobby.

The question of a poor anamnesis or slipshod examination requires no discussion. By ignorance of certain pathologic complexes we mean clinical pictures—by no means rarely encountered—which are not given sufficient emphasis in the ordinary text-book of medicine. As examples of this we might mention the misleading clinical pictures not infrequently produced by the various types of pyelitis, especially in women; by chronic infections of the tonsils, nasopharyngeal tissues, and accessory sinuses, particularly in children; by chronic inflammations of the mediastinal lymph-nodes, also especially in children; by chronic appendicitis and chronic gall-bladder pathology. These are but a few of the many conditions falling into the category under discussion. In their text-book settings they are practically lost as entities; it is only in more or less monographic descriptions that their daily diagnostic importance is emphasized. A thorough understanding of only these few examples of subjects insufficiently discussed in the medical text-books will serve to clear up many otherwise unexplained fevers in the case of pyelitis and of chronic infection of the tonsils, nasopharynx, sinuses, and mediastinal lymph-nodes; and many digestive disturbances in the case of gall-bladder and appendix pathology.

The fourth cause of diagnostic error—a lack of insistence upon the typical—may be illustrated by the adherence to a diagnosis of typhoid fever with a persistently negative Widal reaction or in the presence of an unexplained leukocytosis.

The fifth subhead—too great a dependence upon the laboratory side at the expense of experience and good judgment—stands without further comment. The riding of a hobby—the last point—is also clear enough. Too many doctors, both general practitioners and specialists, are prone to see in a case not otherwise clear a condition upon which their enthusiasm for the moment may be centered. The avoidance of these six faults

makes enormously for correct diagnosis and directly for the prevention of periods of emergency.

A correct diagnosis represents, then, the first and most important step in the treatment of a medical emergency. Reduced to simpler terms, every medical case is a medical emergency because it demands first of all a correct diagnosis. Illustrations of this truth by the score will occur to every one of you, and we shall accordingly not permit ourselves to linger at any great length on this phase of the subject. But how many "colds," bronchitides, and other respiratory infections are regarded as simple affairs because of infrequent or careless chest examination, when they are really pneumonias, the patient being allowed to do things that one ill of pneumonia would never be permitted to do. This is one of the commonest emergencies of daily practice and one fraught with the gravest consequences. Careful nursing, digitalis, venesection, oxygen, or what not may never be able to undo what a slipshod diagnosis might have prevented.

Again, a child with vague rheumatic pains, a slight jerkiness in his movements, perhaps twitching of one or more muscles of the face, is allowed to play about with some such diagnosis as nervousness or habit spasm. In reality, an undeveloped chorea is present, and because unrecognized permits the development of a permanent cardiopathy. Or nervousness, a change in disposition, crying spells, palpitation are set down as neurasthenia, and a crucial period in the diagnosis of a hyperthyroidism is allowed to slip by.

We might multiply indefinitely illustrations of diagnostic undervaluations of conditions with serious possibilities; those mentioned, however, perhaps place sufficient emphasis upon the emergency resulting from an incorrect diagnosis of so-called minor ailments.

Although our theme is essentially one of medical emergencies, we cannot proceed far in the matter of diagnostic emergencies without encroaching upon the field of surgery, the so-called Grenzgebiete. Here the question is largely whether surgery is indicated or not. Too many pneumonias are placed in the sur-

geon's hands with a diagnosis of appendicitis; too many tabetics with gastric crises are made surgical cases because of incomplete examination; too many cases of coronary or abdominal sclerosis are operated upon for gall-bladder disease. Lead-poisoning accounts for many cases of ruptured gastric or duodenal ulcer, acute pancreatitis, etc.; congested liver incidental to cardiac decompensation is too often called peptic ulcer or gall-bladder disease.

All of these are emergencies of diagnosis, and represent a few examples of conditions falling into the borderline state between medicine and surgery.

In this rather loosely assembled presentation you will allow us to pick out two conditions in particular which if not diagnosed early, or, on the other hand, if diagnosed when not present, open the road to limitless future trouble. We refer to tuberculosis and syphilis. The physician into whose hands falls an early case of pulmonary tuberculosis or syphilis is confronting a real emergency, for upon his correct interpretation may depend the life of the patient or, at least, the prevention of irreparable damage. And you will recall that an emergency is just so defined—*i. e.*, an event demanding immediate action to save life or to prevent irreparable damage. Early diagnosis in tuberculosis or syphilis is likely to mean early cure; a delayed diagnosis may mean death or such irreparable damage as is seen in the later stages of consumption, or in the case of syphilis, tabes, general paralysis, aneurysm, etc.

Almost as serious is a faulty diagnosis of syphilis or tuberculosis. It is far easier to make than to unmake such a diagnosis. If in the past a physician has treated it as such, you, seeing the patient at a later day, may find it well nigh impossible to undo the damage done. The havoc here is likely to be irreparable, even though it be largely on the mental and not on the physical side.

The treatment of syphilis and tuberculosis also have their emergency aspects, not so much from the point of view of immediate action as from that of thorough and sufficiently prolonged management. In the case of tuberculosis, once the

diagnosis is established, plans must be drawn for a thorough course of treatment based on the fundamentals of rest, good air, and good food; in the case of syphilis tried remedies must be pushed and continued over a sufficient length of time to ensure results. The emergency is slighted if proprietary forms of mercury are used on the strength of glowing advertisements and if patients are not warned that years of treatment are necessary. The use of sodium cacodylate as fulfilling the demands of treatment is strongly to be condemned.

Medical emergencies arise also in the way of prevention. As examples of this we might mention smallpox, typhoid, and possibly diphtheria and whooping-cough. Vaccination against smallpox is, so far as we know, absolute protection against the disease; in other words, the emergency of facing an actual case of variola should rarely, if ever, arise. It is likely, further, that vaccination against typhoid fever, if properly performed, means immunity for a variable number of years. The experience of several wars seems to allow this conclusion. As to toxin-antitoxin in diphtheria, the story is still not complete, though the experimental work is very promising: while in the case of pertussis the reports are too conflicting to permit of a conclusion, though here, too, the experience of certain observers have been very encouraging. The prevention of any of these diseases, it goes without saying, means the wiping out of an incalculably large number of potential emergencies.

Prophylaxis tends to reduce emergency conditions also in the presence of disease actually present. Examples of this are legion: the proper care of measles to prevent pulmonary complications; measures to prevent renal damage in scarlet fever; frequent examination of the urine in cases of syphilis under mercurial management; sufficiently prolonged bed rest in rheumatic fever and chorea to lessen the tendency to endocardial damage, and in diphtheria to prevent paralyses. Very few diseases, in fact, are so free from possible serious complications that efficient measures during their actual course are not in order to guard against these complications.

The emergency features in the treatment of tetanus require

special consideration from several aspects. As regards prophylactic measures, in the first place, we feel that dangerous situations cannot infrequently be avoided by a better knowledge of how and when to carry out immunizing measures. Tetanus antitoxin, in a preventive way, is given too seldom rather than too often; unhappy clinical experience proves this. Any break in the surface covering of the body involving contact with the soil demands an immediate immunizing dose of antitoxin. In the doubtful case, if previous serum injections raise the question of anaphylaxis, the serum should be injected in the recognized manner of preventing this perhaps overrated reaction. One immunizing dose is not adequate, and, as prescribed by the medical corps in the recent war, a second dose should be injected within a period of ten days.

The emergency of tetanus actually developed must be met promptly and squarely. Haphazard subcutaneous or intramuscular injections no longer answer the requirements; repeated intravenous and intraspinous introduction of serum—as well as subcutaneous or intramuscular—must be carried out, depending on the severity of the case and the response to treatment. Statistics show that intensive therapy of this type has materially lowered the death-rate from this disease. We need hardly add that the usual surgical treatment of the wound is still another phase of the emergency.

A more general understanding of the importance of early diagnosis of diphtheria has materially lessened the number of emergency types of this condition we are called upon to see. However, a certain number of ominous cases still present themselves, and in these the intravenous introduction of serum must be pushed together with the usual subcutaneous or intramuscular injections.

A somewhat similar situation is encountered in epidemic cerebrospinal meningitis. Recent experience has demonstrated that the intraspinal method must be reinforced by the intravenous to obtain the highest percentage of cures.

If we return now to our theme in its narrow sense, we find that there is not a great deal deserving special comment. We

have gone through a comprehensive text-book of medicine and selected the conditions not already discussed in some detail that require prompt measures to save life. Unfortunately, at the present time at least, many of the more serious medical diseases are of more interest from a diagnostic than from a therapeutic standpoint.

The list is as follows:

The poisonings.

Severe forms of malaria requiring intravenous quinin.

Diabetic coma.

Peptic ulcer: perforation and hemorrhage.

Typhoid fever: perforation, hemorrhage, and certain other complications.

Organic stenosis of the pylorus.

Edema of the larynx.

Bronchial asthma.

Large accumulation of fluid in the pleural cavities.

Anuria and hematuria.

Uremia.

Purpuras of the type of morbus maculosus.

Large pericardial effusions.

Broken cardiac compensation.

Pulmonary edema.

Angina pectoris.

Acute hyperthyroidism.

Cerebral hemorrhage, embolism, and thrombosis.

Certain brain and spinal cord tumors accessible to surgery.

No doubt this list is incomplete, but it is sufficiently inclusive for our purpose. It shows, namely, what we have attempted to demonstrate earlier in our talk, that the actual management of these emergency situations is completely overshadowed in importance by the prophylactic and diagnostic aspects of the same condition.

To illustrate this contention let us discuss several of these emergency conditions more in detail. Confronted with an apparently life-threatening asthmatic attack, we ought not to content ourselves with one or more injections of epinephrin;

the situation demands that we make an attempt to determine whether the asthma is of the true bronchial type, in which case epinephrin is strictly indicated, or whether it is of cardiac origin, demanding digitalis and possibly venesection or morphin, or whether it is renal or uremic, calling for cardiac treatment (if the uremia is of the cardiac type) or eliminative treatment (if the uremic seems to be purely renal in origin).

Or, in large pleural or pericardial effusions, we must first satisfy ourselves of the diagnostic possibilities, both as to the actual presence of fluid as against other conditions producing similar clinical pictures and physical findings, and as to the amount of fluid present, before we take recourse to the needle.

Indeed, the further we sift the matter, the fewer medical conditions do we encounter in which immediate treatment cannot give the right of way to a rapid, clean-cut effort at diagnosis. And once a diagnosis is tentatively made, the actual treatment is in the hands of every physician—the accepted text-books of medicine contain all that is necessary for us to know.

And this is the reason why we have felt it essential in a talk on the treatment of medical emergencies to devote practically all of our time to the matters of diagnosis and prevention and very little to the actual medical management of such emergencies.

CLINIC OF DR. DON C. SUTTON

COOK COUNTY HOSPITAL

HAY-FEVER

WHILE hay-fever occurs mainly during the summer months, and therefore makes it impossible to present cases at this time, I shall take this opportunity for its discussion, first, because of its importance, and second, because of the similarity in etiology of hay-fever and asthma.

The usual conception that hay-fever occurs only during the summer months must be modified to include many cases that have been classed as hyperesthetic rhinitis and occur throughout the year.

The symptoms of hay-fever are intumescence of the nasal mucosa, with profuse watery discharge, and extreme irritability resulting in incessant sneezing. Usually the conjunctivæ are markedly congested and lacrimation much increased. These symptoms are the result of a reaction apparently of the nature of a local anaphylaxis to a specific foreign protein.

Since the most common protein causing this disease is the pollen of certain flowers, the name hay-fever and the seasonal occurrence are easily accounted for. Hay-fever, then, may be separated into groups varying with the season of the year and groups separated by the causative proteins.

I. *Seasonal Group.*—This includes mainly pollens of spring, summer, and fall flowers.

A. Pollens of early spring, giving rise to symptoms usually known as rose fever—dandelion, willow, maple, poplar, and occasionally other trees and flowers.

B. Pollens of late spring and summer—June grass, red top, orchard grass, timothy, daisy.

C. Pollens of late summer and fall—ragweed (giant and dwarf

varieties), corn (maize), sunflower, daisy, goldenrod. Any plant with a pollen that floats in the air may be an occasional cause.

D. Bacteria may be a seasonal cause.

E. Animal emanations (may be seasonal when contact is only seasonal).

F. Foods (either inhalation during preparation or after eating).

II. *Non-seasonal Group*:

A. Animal emanations (hair or dander):

Horse,	Goose feathers,
Dog,	Chicken feathers,
Cat,	Duck feathers,
Rabbit,	Furs.

B. Foods.

C. Bacteria.

D. Dusts containing proteins.

In this locality the most prevalent of the seasonal group is the third, namely, during August and September. For instance, in an analysis of a group of cases of seasonal hay-fever ragweed was causative in 85 cases, corn in 5, goldenrod in 3, timothy in 3, red top in 2, sunflower in 3, and dandelion in 1. At times cases are sensitive to one or more pollens throughout two or more groups.

The non-seasonal group, of course, develops hay-fever at any time of exposure.

Exposure to animal emanation is often through articles made from furs and feathers. Occasionally exposure may be indirect, as in the case of a child seen recently whose hay-fever and asthma were due to horse dander carried upon the clothing of the father, a hostler.

Dusts include a variety of occupational dusts, as fur and garment making, bakers, wood workers, and, not the least, the housewife is often sensitive to dusts arising in the house.

Face powders containing orris root or rice powder are a frequent cause.

Bacteria are causative from foci in the sinuses or upon the nasal mucosa.

When the close relationship of asthma to this group is recalled, it is readily seen that the determination of the causative protein is of extreme importance. Fortunately, if the offending protein comes in contact with the cells of the skin, a local reaction results, namely, a wheal surrounded with red areola. This reaction occurs when a solution of the protein is injected intracutaneously or when applied to an incision through the epidermis. Both methods are used in determining the cause.

(Here the test is demonstrated upon a student sensitive to pollens.)

At times all tests fail to reveal the cause. This may be due, first, to our own ignorance; second, an anti-anaphylactic state; third, to the fact that preparations deteriorate so that apparently negative results may become positive when fresh extracts are used.

The treatment is specific in that an endeavor is made to desensitize the individual to the causative protein. This method offers encouraging results in the cases due to pollens, and less so in those resulting from other causes. Of course, the removal of the cause results in prompt cure.

The treatment of pollen sensitization consists of subcutaneous injection of aqueous extracts, beginning with small doses and carrying the desensitization over a long period of time with gradually increasing doses, and ending at the time of flowering of the causative plant. In my own work I have continued to prepare my own extracts, not that they are superior to other preparations, but mainly because fresh extracts are then used. These extracts are prepared in two ways: One, the method of I. Chandler Walker, is a 1 per cent. extract of the pollen in normal salt solution and sufficient alcohol to make a 12.5 per cent. alcoholic solution. Dilutions are then made containing 1 part of pollen to 20,000 normal salt, .1 : 10,000, 1 : 5000, 1 : 3000, 1 : 1000, 1 : 500, 1 : 250, and 1 : 100.

Second, I have prepared a suspension of pollen in olive oil. The pollen is ground in an agate mortar, suspended in oil, and sufficient phenol added to make ½ per cent. in the 1 : 100 dilution. Dilutions are made of 1 : 5000, 1 : 3000, 1 : 2000, 1 : 1000,

and 1 : 500. The olive oil delays absorption and the suspension adds the proteins that are left in the residue of water extracts, also the slow absorption allows the use of larger doses and decreases the dangers of severe reactions.

The initial dose of the extracts is determined by making skin tests, using all the dilutions of pollen to which the patient is sensitive. The initial dose is then 0.2 c.c. of the next higher dilution, *e. g.*, the least positive reaction shown is 1 : 3000, then the first dose is 0.2 c.c. of 1 : 5000. The usual interval between doses is five days, but by depending upon the severity and duration of the reaction at the site of injection doses may be given daily to three-day intervals. The dose is increased in 0.2 c.c. stages, using the next lower dilution when the amount of pollen in the dose is equal to that contained in 0.2 c.c. of the next lower dilution. The final dosage varies from 0.5 c.c. of 1 : 250 to 0.5 of 1 : 100, this dose being maintained at five-day intervals until season begins.

When dilutions lower than 1 : 1000 are reached general reactions may at times occur. These are an attack of typical hay-fever; often at the same time there is intense redness and itching of the skin or even urticaria, and in more severe reactions asthmatic attacks. These reactions are promptly relieved by 10 minims of adrenalin (1 : 1000), but every precaution should be taken to avoid them. It has been observed at times that, contrary to the usual effect, there is a partial loss of the desensitization already acquired. There have been no general reactions with the use of the oil suspension.

The results obtained by these two pollen preparations have been: For *salt extract* varying from year to year 35 to 50 per cent. have been completely relieved for the season; 50 to 75 per cent. have been markedly improved. This improvement consists of either a mild hay-fever through the season, or more often the symptoms come on two to three weeks later than usual, are of usual severity for one to two weeks, and then followed by almost complete relief for the remainder of the season. Finally there is 10 to 25 per cent. that are entirely unaffected.

For the *oil suspension* in a small series the results have

been better, namely, 80 per cent. completely relieved and 20 per cent. moderately improved, to complete failure.

Auld, of England, has had excellent results with the use of a 5 per cent. solution of peptone intravenously. He uses Armour's No. 2 peptone, which is an impure preparation containing many of the higher protein fractions. He recommends that it be given intravenously in doses from 0.2 to 1.2 c.c. I have used this solution for three years. The first year 88 per cent. were completely relieved, the last two years 100 per cent. were complete failures. Each year the preparation was made from a different package of Armour's No. 2 peptone. That used the first year caused chills in doses of from 0.8 to 1 c.c. The other years there were no reactions from much larger doses. I therefore concluded the benefit derived from the first preparation was mainly that of the impurities it contained and not the peptone. Accordingly, typhoid bacilli were used to produce a foreign protein reaction in a number of patients during the last hay-fever season. Sufficient typhoid bacilli were injected intravenously to cause a mild chill and slight fever reaction. The dose is uncertain because various cultures vary in the amount required to produce a chill. The preparation we used produced an ideal reaction in doses of 8,000,000 to 10,000,000.

The cases treated by this method were treated during the period of symptoms, with the following results:

First, immediate marked relief within twenty-four hours of first dose.

Second, 80 per cent. were completely relieved by three or four doses.

Third, 20 per cent. were more than 75 per cent. improved after three or four doses.

We have since used typhoid in the treatment of horse dander asthma, and other anaphylactic asthmas of unknown origin, with excellent results. Unfortunately, any method of desensitization results only in temporary immunity, so that these cases must be treated every season.

We may then summarize:

I. That at present the best method of treatment for hay-

fever is an endeavor to desensitize to the specific protein or pollen, keeping in mind that care in the use either of the specific or non-specific protein must be used to avoid severe reactions which may at times be dangerous or even fatal.

II. That the effect of non-specific protein does not as yet warrant its use in treatment, but calls attention to the question of difference in action between specific and non-specific reactions. I feel that the specific protein acts in the same manner as the non-specific foreign protein, a point also brought out by the work of Novy.

CLINIC OF DR. J. P. GREENHILL

CHICAGO LYING-IN HOSPITAL

THE VALUE OF *x*-RAY IN OBSTETRICS

THE field for roentgenography in obstetrics is still a virgin one and has great possibilities. At the Chicago Lying-in Hospital during the last few years we have made very frequent use of the *x*-ray to aid us in our obstetric diagnoses. We utilize the *x*-ray to verify opinions regarding pelves, such as contraction (absolute and relative), deformities, fractures, separation of the symphysis pubis, and healing of pubiotomized pelves. We also corroborate suspicions of fetal deformities *in utero*, such as hydrocephalus and anencephalus, multiple pregnancy, early pregnancy, pseudocyesis, abnormal presentation and position, and fetal death. After delivery we verify fractures of the fetal bones and have demonstrated also such fetal anomalies as dextrocardia and occult spina bifida. We have found early pregnancy where some physicians have thought fibroids were present.

The use of the *x*-ray in obstetrics is not, however, limited to the maternal abdominal and pelvic regions, but is also utilized for examination of the teeth, sinuses, gall-bladder, etc., to detect focal infections.

Methods have been elaborated to accurately measure the pelvis by means of the *x*-ray. However, until the head can be measured in the same way and the exact cephalopelvic disproportion determined, pelvic measurements *per se* are of limited value.

I shall show you today a series of cases which demonstrate how we have been aided in obstetrics by the *x*-ray. The first case under consideration presents the following history:

Case I. Cephalopelvic Disproportion.—Mrs. M. K., No. 25,894, a septipara, was admitted to the hospital in labor. She was very short in stature, being only 4 feet, 10 inches in height, and weighed 157 pounds. Her general physical examination was negative except for unusually large and numerous varicose

Fig. 93.—Cephalopelvic disproportion. Cesarean section. Weight of baby, 4555 gm. (10 pounds, 1 ounce).

veins on the legs, thighs and labia, and in the vagina. Her external pelvic measurements were 23, 26.5, 31.5, and 20. There was a marked sacral promontory and the diagonal conjugate was 11.5 cm. Her obstetric history is of considerable interest. There had been one miscarriage and six full-term pregnancies. All but one of the six labors had to be terminated instrumentally

(forceps). Only two babies were born alive. One of the latter was delivered by Dr. DeLee, who did a difficult high forceps operation. The baby weighed 3401 grams (7½ pounds).

At the time of the present admission abdominal examination revealed a large baby and the head was overrriding the pubis; *x*-Ray pictures were taken, and this (Fig. 93) is one of them. You will observe the small, flat pelvis, and the disproportionately large head. You will also note that the head is at O. L. P. By the time the *x*-ray pictures were taken the patient had been in labor twenty-eight hours and the bag of waters had been ruptured all this time. Since the head was still riding over the pubic bones it was thought that the patient had had a sufficiently long test of labor, and had demonstrated her inability to deliver the baby. The cervix at this time was dilated 8 cm. and was thick. I therefore did a low cervical cesarean section and at the same time sterilized the patient (at her own request). The baby weighed 4555 grams (10 pounds, 1 ounce), which confirmed the *x*-ray findings that the head could not go through the patient's pelvis. (Recall that Dr. DeLee delivered with difficulty by high forceps a baby weighing 3401 grams.)

Case II. Hydrocephalus.—The second case is that of a tertipara, Mrs. H. N., No. 25,812, who had had two normal labors. The babies weighed 2948 grams (6½ pounds) and 3288 grams (7¼ pounds) respectively. The general physical examination was negative and the pelvic measurements were normal. The patient had been sent to the hospital by her physician for cesarean section because he believed the child was too large to go through the pelvis.

On admission I examined the patient and made a diagnosis of a small baby, approximately 6 pounds, with a very large hydrocephalic head. The difference of opinion was settled by the *x*-ray which I am now showing you (Fig. 94). You will observe the outline of the pelvis and that of the head, which is enormous. The head overrides the pubis, the sutures are wide, and the fontanels unusually large. Compare this head with a normal size head or even with the head of the baby just shown (Fig. 93).

which weighed 10 pounds, 1 ounce, and you will appreciate how much larger is the hydrocephalic head. Labor was induced by means of castor oil and quinin. It was decided to do a version and extraction with craniotomy on the after-coming head when

Fig. 94.—Normal pelvis. Hydrocephalus. Perforation and extraction.

dilatation was complete. If during the extraction we found a spina bifida we would have further evidence in favor of a hydrocephalus. However, an unusual occurrence made version and extraction unnecessary. The head, which was large and

cystic and had very thin bones, was forced down to the pelvic floor by slow uterine and abdominal contractions. The head during a pain felt as tense as a football, whereas the tension was much less in between pains. The shape of the head during a pain resembled that of a dumb-bell, with one half, the smaller, in the true pelvis and the other half, the larger, in the false pelvis. A trocar was inserted into the anterior fontanel and about 1½ liters of straw-colored fluid escaped. The base of the brain was destroyed and the child was readily extracted by inserting a finger in the skull. After delivery the skull bones were found to be unusually thin and soft. This accounts for the faint shadow on the *x*-ray plate. No spinal bifida was seen in the baby. However, when an *x*-ray was taken an occult spina bifida was found. In this case, therefore, the *x*-ray helped us twice.

Case III. Tuberculosis of the Hip.—The third *x*-ray plate shows, as you see, an oblique deformity of the pelvis (Fig. 95). This patient, Mrs. E. S., No. 11,928, had quite a marked disturbance in gait. The history and physical examination pointed to a tuberculous infection of the left hip. Internal examination revealed a very prominent ischial spine on the left side. The *x*-ray was taken to determine how much deformity of the pelvis there was, with a view to determining the chances of delivery *per via naturalis*. As you see, the pelvic cavity is roomy. Note the oblique deformity on the right side, the prominent ischial spine on the left, and the tilting of the pelvis on the left. Observe also the differences in the two femora as regards caput, position, and thickness. In view of the spaciousness of the pelvis, as shown on the *x*-ray plate, the patient was permitted to continue in labor, and she delivered a large baby weighing 4365 grams (9 pounds, 10 ounces) spontaneously. The entire labor lasted only five hours and thirteen minutes.

Case IV. Pubiotomy.—The *x*-ray plate (Fig. 96) of Mrs. C. U., No. 21,555 (Dr. Cornell), which I now hold, shows a pubiotomized pelvis. The pubiotomy was done elsewhere and a

baby weighing 3827 grams (8 pounds, 7 ounces) was delivered. The patient remained in the hospital for three weeks postpartum and was unable to walk well for two months after this. During the present, her second pregnancy, the patient felt fairly comfortable. There was no disturbance in gait. The external

Fig. 95.—Oblique contraction of the pelvis; tuberculosis of the left hip. Roomy pelvis. Spontaneous delivery of baby weighing 4365 gm. (9 pounds, 10 ounces).

pelvic measurements were 25, 26.5, 29, and 18, and the diagonal conjugate measured 11 cm. The patient had an easy labor and delivered spontaneously a baby weighing 3740 grams (8 pounds, 4 ounces). You will note the wide separation on the right side of the two ends of the pubic bone which had been cut. The pelvic cavity was enlarged, and the second baby, which weighed

almost as much as the first, was delivered very quickly, labor lasting only four and a half hours.

Case V. Separation of the Symphysis Pubis.—Mrs. J. M., No. 22,249, a primipara, rather short and stout, was seen in our

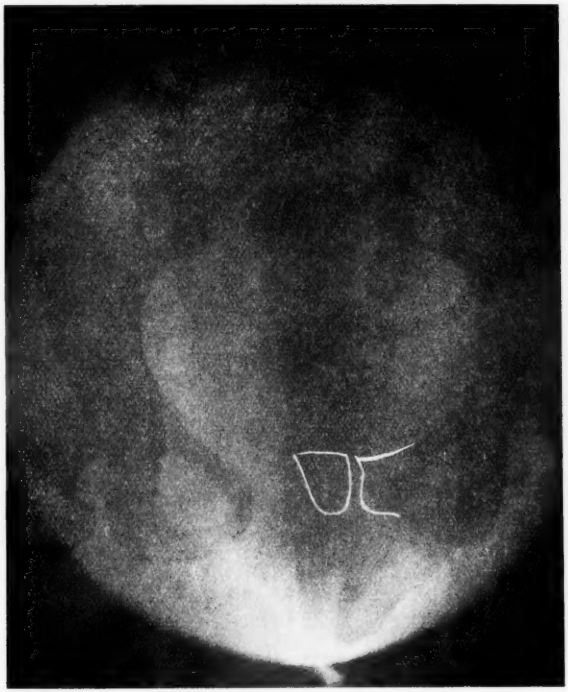

Fig. 96.—Pubiotomized pelvis. Easy spontaneous delivery in subsequent labor.

prenatal clinic. She was extremely nervous and a vaginal examination was made with extreme difficulty. The vagina was very tight and would barely admit one finger. The external pelvic measurements were 22, 26, 34, 20.5, and the pelvis was of a masculine type. The pubic arch was unusually narrow.

Labor was very prolonged, the first stage lasting fifty-three and a half hours; but after one hour and forty minutes of second-stage pains the patient delivered spontaneously a large baby weighing 3535 grams (7 pounds, 13 ounces). Two days after delivery the patient complained of severe pain over the symphysis pubis. She refused to permit palpation of the symphysis,

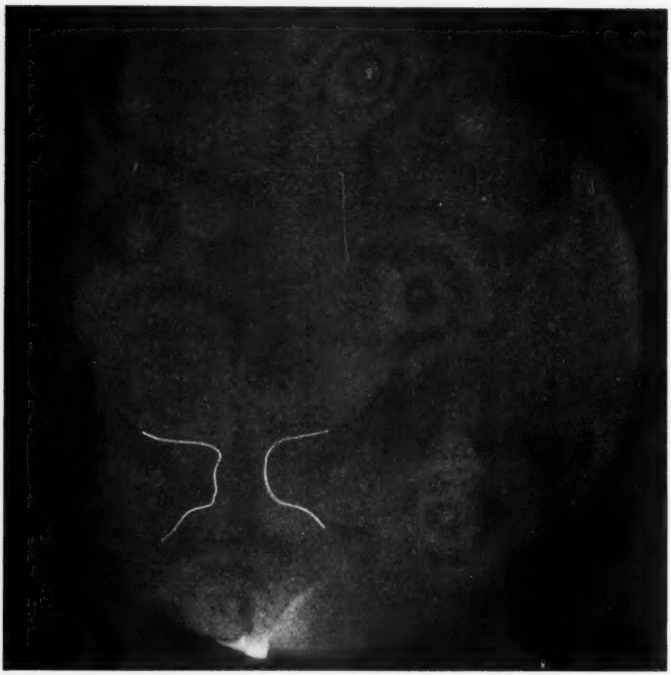

Fig. 97.—Spontaneous separation of symphysis pubis.

and cried out when attempts were made to elevate her on a bedpan or to change her position. A diagnosis of separation of the symphysis pubis was made and verified by *x*-ray, as you see (Fig. 97). The pubic bones are separated for almost 1 inch. The pelvis was strapped with wide pieces of adhesive and the pains gradually diminished. The patient was discharged on the

thirteenth day postpartum, with no disturbance in gait, but still complaining of tenderness in the region of the symphysis. She was examined eight weeks later and, while the bones had come together, the symphysis was still tender.

Fig. 98.—Fracture of left pelvic bone after midforceps delivery.

Case VI. Fracture of the Pubic Bone.—A somewhat similar case was that of Mrs. G. B., No. 17,861 (Dr. DeLee). She was a primipara and had a large roomy pelvis. The child, which weighed 3920 grams (8 pounds, 10 ounces), was delivered with

some difficulty (midforceps). The day after delivery the patient complained of pain in the symphysis on movement. Examination revealed tenderness, but no separation of the symphysis. An *x*-ray picture was taken and, as you see (Fig. 98), shows a fracture of the left pubic bone at the symphysis. The pelvis was strapped and, while relief was obtained, there was a marked disturbance in gait. A brace was made and this helped considerably, but the patient could not walk properly without the brace for more than a year.

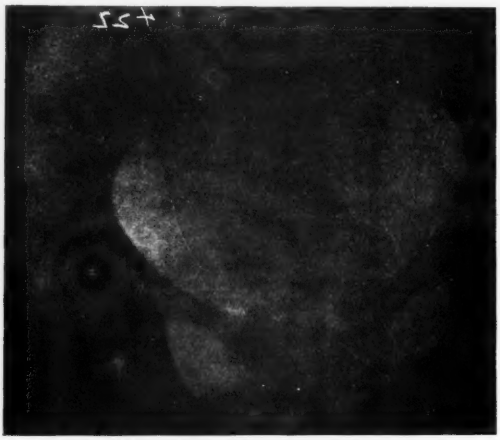

Fig. 99.—Breech presentation. Premature baby.

The plates which I have thus far shown you have dealt with abnormalities in the pelvis, both absolute and relative. I shall now show you a few plates which were taken to obtain information about the fetus or fetuses.

Case VII. Breech Presentation.—The first plate (Fig. 99), as you see, shows plainly a breech presentation. This picture was taken three weeks from term, as calculated from the patient's menstrual history (Mrs. R. M., No. 26,324). However, as you may see, the fetus is very small and when born (on the same day the *x*-ray plate was taken) weighed only 1720 grams

(3 pounds, 13 ounces) and showed the evidences of prematurity.

Fig. 100.—Triplets. Note the three heads.

Case VIII. Triplets.—The plate which I now hold (Fig. 100) is of considerable interest, for to my knowledge it is the only one showing triplets. The patient, Mrs. F. E., No. 21,024, was

a para II and was first seen in our prenatal clinic when about twenty-seven weeks pregnant. The abdomen, however, was about the size of a full-term twin pregnancy. The patient slept poorly and only in a sitting position because of the orthopnea produced by the pressure of the abdominal contents on the diaphragm and heart. Examination revealed two distinct sets of heart tones. Because of the unusually large abdomen for the period of pregnancy and the finding of at least two separate heart tones, a diagnosis of triplets or twins polyhydramnios was made. An *x*-ray was taken, but only two heads were found. A few days after admission the patient went into labor spontaneously; the first baby, which presented by the breech, was easily extracted. A second bag of water was found and ruptured artificially. The second baby, which presented by the head, was delivered with forceps. A third sac was found and ruptured, and the third baby delivered by the breech. We returned to the *x*-ray plate and after careful examination definitely located a third head. In this case the *x*-ray plate was not at fault, as we first thought, but we were to blame for not having made a very careful examination of the plate.

Case IX. Fetal Death.—Another plate of unusual interest is that of Mrs. D. S., No. 25,500, a quintipara. When about thirty-six weeks pregnant the patient said she suddenly ceased to feel fetal movements. No fetal heart tones could be heard. The uterus was somewhat smaller in size and should have been found in accordance with the calculated date. On vaginal examination the fetal head felt hard. An *x*-ray was taken, but the fetus presented nothing unusual. The patient was told to await labor-pains unless unusual symptoms occurred. Twelve days later the patient returned in labor. No fetal heart tones could be heard. Rectal examination revealed a soft, somewhat macerated head from which the bones overlapped. An *x*-ray was taken (Fig. 101) which shows the characteristics of fetal death as described by Horner and later independently by Spaulding. You will note overlapping of the skull bones, asymmetry of the head, and also wrinkling of the fetal scalp. The

fetus, which was delivered the same day, was badly macerated. This *x*-ray sign of fetal death is of vast importance and, aside from the actual palpation of the macerated skull, is the only positive sign we had of fetal death.

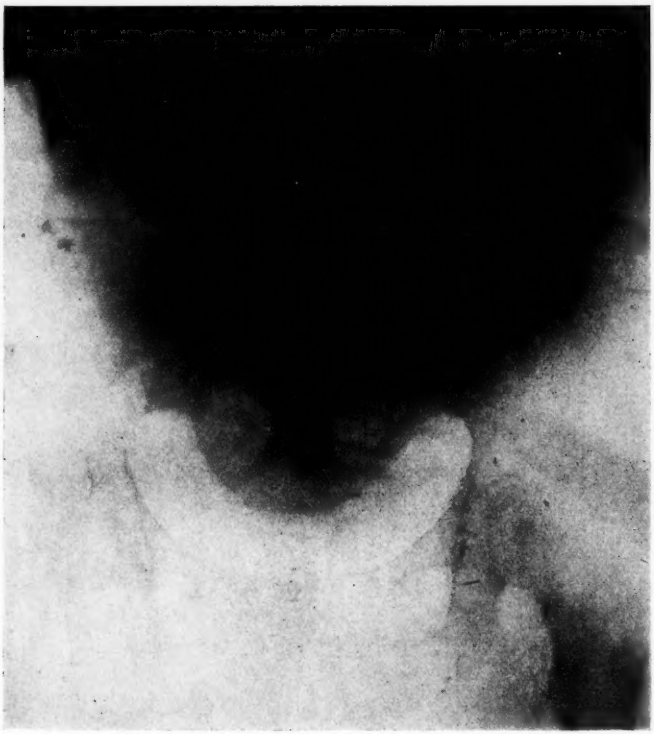

Fig. 101.—*x*-Ray showing fetal death. Note overlapping of bones and wrinkling of scalp.

Case X. Tubal Pregnancy.—The next *x*-ray plate for consideration was taken after the injection of air into the peritoneal cavity. Mrs. R. B., No. 11,845 (Dr. Cornell), had had one miscarriage at three months, after which her uterus was packed. She had had no other pregnancies. Six years before admission she had her appendix removed because it was "a pus appendix."

On August 5, 1921 at 10 P. M. the patient suddenly experienced difficulty in breathing, her abdomen became distended, and there was marked palpitation of the heart. There was also pain in the right lower quadrant. The patient felt nauseated and vomited once. The next day she felt better, but during the following week there was marked lassitude. On the 16th she menstruated, this being the regular time for her menses. The pain during the period was aggravated, being most marked in

Fig. 102.—Pneumoperitoneum. Note enlarged uterus and tubal pregnancy on right side.

the left lower quadrant. The bleeding continued for two weeks, at the end of which time the patient came to the hospital. Abdominal examination revealed tenderness in both lower quadrants. On vaginal examination a mass was found on the right side. The pneumoperitoneogram, as you see (Fig. 102), shows an enlarged uterus with a normal ovary on the left side. On the right side is an elliptical-shaped mass which was diagnosed as an ectopic pregnancy. Operation verified this diagnosis.

Case XI. Chrondrodystrophia Fetalis.—The plate which you now see (Fig. 103) is that of a case of chrondrodystrophia fetalis (Dr. Bloomfield). The child's mother was a primipara, aged thirty-three. The child, which was born alive and lived two hours and forty minutes, weighed 2980 grams (6 pounds,

Fig. 103.—Chondrodystrophia fetalis.

9 ounces) and measured 39.5 cm. in length. The trunk measured 20 cm., while each of the upper extremities was 12.5 cm. long, and each of the lower 10 cm. in length. Note the short bones in the extremities. As a contrast compare this plate with that of a normal size baby (Fig. 104). Observe the difference in

lengths of the bones, especially that of the long bones. Incidentally notice the fracture in the middle third of the left clavicle of the normally proportioned baby.

The other *x*-ray plates I have here demonstrate an anencephalus, an obliquely contracted pelvis of marked degree, a transverse presentation, a face presentation, and the case of bag induction showing the displacement of the fetal head.

Fig. 104.—Normal sized baby, to be compared with Fig. 103. Note fracture at middle third of left clavicle.

The few *x*-ray plates shown will give you an idea of the field of usefulness for the *x*-ray in obstetrics. We do not hesitate to employ the *x*-ray, for we have seen no harmful effects from it on either mother or child. This is probably due to the fact that the exposures are very short.

In cases of placenta prævia where a cesarean section is contemplated *x*-ray pictures should be taken; for, as I recently pointed out (Surgery, Gynecology, and Obstetrics, February,

1923), the association of fetal monsters and deformities with placenta prævia is not an unusual one. Indeed, in all cases where abdominal delivery is contemplated, no matter what the indications, if a definite occiput is not felt or if the head appears abnormally large, it is advisable to have an *x*-ray picture taken before operation.